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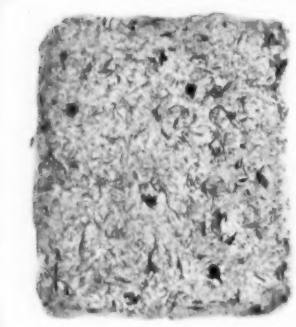

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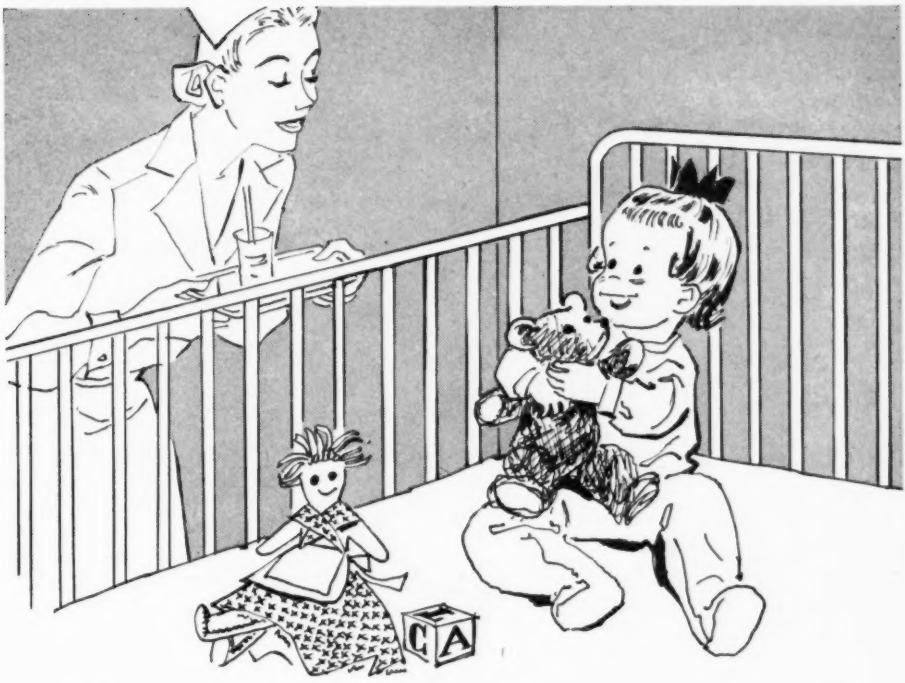

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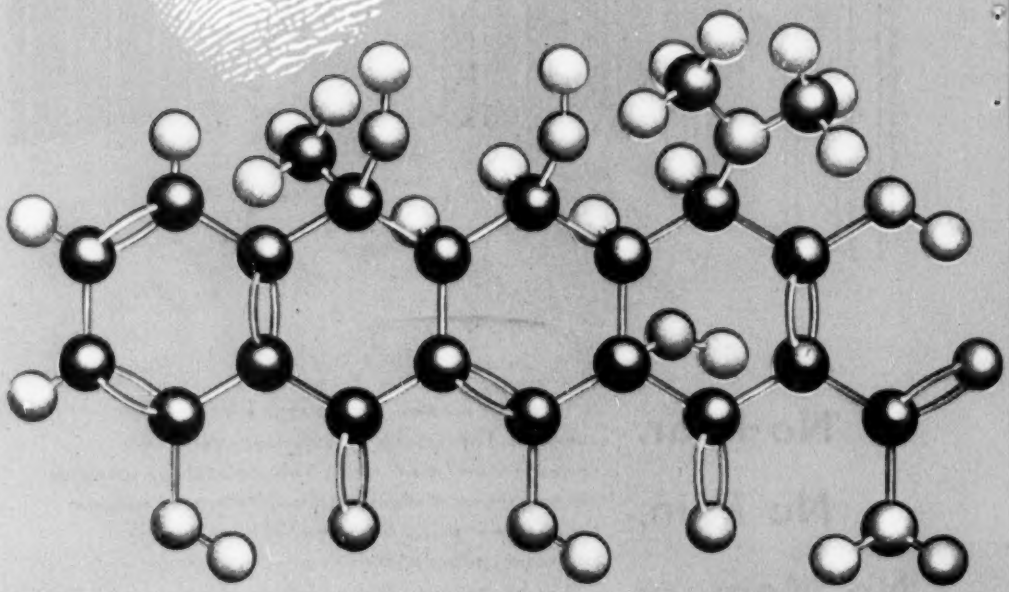

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Editorials

PREOPERATIVE TRANSFUSIONS IN GERIATRIC SURGERY

IN OPERATIONS of election it is now fairly commonplace to correct blood deficits by transfusion before operation rather than to wait and transfuse only in case of circulatory impairment during or after operation. Pronounced loss of weight leads to a deficit in both the red cell mass and the total circulating plasma proteins, a conclusion based on many careful blood volume measurements which have shown considerable reductions despite normal routine laboratory reports. Indeed, many surgeons do not hesitate to use blood transfusions in preparing severely malnourished patients even if the red counts and hemoglobin concentrations are normal.

That advancing age in adult surgical patients may be associated with a blood deficit is suggested from the blood volume studies by Beling and his co-workers in a recent number of *Geriatrics*.[1] These workers employed the standard Evans blue (T-1824) and hematocrit technic. In a preliminary survey apparently in an unselected group of patients, the values for 100 individuals above 60 were compared with those for 100 below 60 years of age. The figures in the former group were lower than in the latter, e. g., the plasma volume in milliliters per kilogram in the older group had a mean value of 39.6, as compared with 42.8 in the younger group, the standard error being 0.35. Evaluation of these data, however, is difficult because even in normal healthy individuals the blood volume per kilogram of body weight varies tremendously with the size of the skeleton and particularly with the mass of relatively inert adipose tissue. It might perhaps have been better for these workers to have used body surface by measuring height and weight, rather than body weight alone, or still better to have correlated the findings with the degree of obesity or with recent weight loss. Unfortunately, there have been few studies by others on the influence of advancing age on blood volume. For example, in normal human adults, Gibson and Evans [2] found a tendency for the blood volume to fall with advancing age, whereas Cohn and Shock [3] found very little significant vari-

1. Beling, C. A.; Bosch, D. T., and Carter, O. B.: Blood Volume in Geriatric Surgery, Geriatrics **7**:179, 1952.

2. Gibson, J. G., and Evans, W. A., Jr.: Clinical Studies of the Blood Volume: II. The Relation of Plasma and Total Blood Volume to Venous Pressure, Blood Velocity Rate, Physical Measurements, Age and Sex in 90 Normal Humans, J. Clin. Invest. **16**:317, 1937.

3. Cohn, J. E., and Shock, N. W.: Blood Volume Studies in Middle-Aged and Elderly Males, Am. J. Med. Sc. **217**:388, 1949.

ation. The number of patients studied, however, was not large. Moreover, Parsons, Williams, and Forsythe [4] failed to confirm the findings of Beling and co-workers as to the influence of age on blood volume in a series of surgical patients.

As already mentioned, fat is a relatively inert tissue and contributes very little to the blood volume, while at the same time increasing body weight. That the amount of adipose tissue increases with advancing age was shown by Brozeck and Keys [5] who found the body fat expressed as percentage of body weight was 9.8 in a group aged 19 to 25 years, as compared with 21% in an older group aged 45 to 55 years. The body weight in both groups was the same. From these studies, one would expect a lower total blood volume per kilo of body weight in older individuals—not because they were old, but because they have a larger amount of tissue fat.

In view of this important finding, it is clear that the use of present methods of blood volume measurement uncritically applied may lead to difficulty. To illustrate the inherent inaccuracies in using "mean" or average data one need only cite the wide variations of between 32.2 and 62.4 milliliters per kilogram in the plasma volume of a series of normal individuals recorded by Cohn and Shock.[3]

Of more immediate surgical interest was the comparison by Beling and co-workers of a group of 100 elderly surgical patients given enough blood before operation to restore their "calculated" blood volume to a "normal mean" of 85 milliliters per kilogram with an apparently comparable group of 190 elderly patients given blood based purely upon bedside indications. The actual amount of blood given averaged 1,500 ml. in the first as compared with 750 ml. in the second group.[6] The postoperative mortality was 8% in the first as compared with a death rate of 17.4% in the second group.

Analysis of the causes of death in the two groups is of special interest in view of the supposed danger to the cardiovascular system of giving blood transfusions to elderly patients. This fear was not substantiated in this study. Indeed, the evidence suggests that the circulation was improved by the use of large amounts of blood. For example, there were no deaths from postoperative shock or cardiac failure in the 100 older patients operated upon after routine preoperative blood replacement. By contrast, among the 190 patients given blood for the usual bedside indications, there were 10 deaths listed as due to cardiac failure and 3 to postoperative shock. On the other hand, it was interesting to note that of those given preoperative transfusions, pulmonary embolism was listed as the cause of death in 4 patients (4%), but of the control group, in only one (0.5%).

Blood volume studies are, of course, subject to many adverse criticisms, some of which have already been discussed. Despite these objections, the studies of Beling and co-workers are valuable in that they suggest a possible means of reducing the mortality in geriatric surgery, especially by eliminating the traditional fear that

4. Parsons, W. H.; Williams, W. T., and Forsythe, L. J.: The Clinical Significance of Blood Volume Determinations, Ann. Surg. **135**:791, 1952.

5. Brozek, J., and Keys, A.: Limitation of the "Normal" Weight as Criterion of Normality, Science **112**:788, 1950.

6. Bosch, D. T.; Islauri, A.; Tau, C. T. C., and Beling, C. A.: The Elderly Patient: An Analysis of 500 Consecutive Cases of Patients 60 Years of Age or Older, A. M. A. Arch. Surg. **64**:269, 1952.

older patients tolerate blood transfusions poorly. What needs to be emphasized is the special importance of recognizing and correcting deficits in the older group because deficits are much more hazardous in operating on older patients than they are in the younger group. At the same time, mistakes in correcting deficits are more serious in older than in younger patients. A note of caution is therefore in order lest undue consideration be given laboratory data just because they are expressed in exact numerical terms. Clinical judgment tempered by sound physiologic knowledge is still a useful guide to replacement therapy. Moreover, the danger of overtreatment must be emphasized in view of the increasing availability of blood banks. Unnecessary and excessive blood transfusions must be counted as more harmful than none at all. On the other hand, exact replacement is better than haphazard or no replacement. Nevertheless, the findings of Beling and co-workers add further evidence of a simple but important principle of therapy which cannot be too often repeated—needed biochemical replacement should be early rather than late, or to quote Owen Feltham, "Prevention is the best bridle."

ROBERT ELMAN, M.D.

THE HERNIA PROBLEM

IT MAY come as a surprise to learn that according to the United States mortality tables hernia (exclusive of intestinal obstruction) in 1948 is listed as responsible for about 5,000 deaths, a figure which exceeded the death rate for acute appendicitis in that year. Moreover, this total mortality has remained nearly constant for the past two decades, a period during which the mortality from acute appendicitis has been cut sharply to about one-third. These fatalities are, of course, not due to the hernia itself, but to its complications, i. e., strangulation or obstruction, which are readily prevented by an adequate, properly executed operation. Inasmuch as hernia should be readily recognized and effectively repaired with practically no risk, there is really no excuse for the high death rate. One aspect of the problem of hernia, therefore, is the need for a drastic reduction of mortality by the preventive procedure of herniotomy.

The problem of hernia is larger than this when one realizes that the number of individuals in this country with unrepaired hernias has been estimated by the United States Public Health Service as about 800,000, a figure which correlates well with the sale of trusses, which is said to exceed 1,000,000 per year. From the medical point of view, this situation offers a sharp therapeutic challenge. From the patient's point of view it means harboring a disease which always carries a threat to life and with which normal health and activity is relatively difficult or impossible. Neither of these two problems can be completely eliminated by the use of even a well-fitted truss. Why, then, are there so many unrepaired hernias? The fault lies partly with patients in that they seem to fear operation (or recurrence), or have erroneously associated operation with loss or diminution of sexual power. Yet it is a common experience for surgeons to hear patients express pleasant surprise after a successful herniotomy in the following terms: "If I had known the operation was so simple and effective, I would not have carried this thing around for so many years." Lack of finances is sometimes used as an excuse for postponing operation, although with the increasing use of various insurance plans this reason is less and

less valid. Moreover, in the case of indigent patients provision can usually be made for meeting the cost. Even if there is no local agency there has been provided throughout the continental United States by means of the Physical Restoration Extension (Public Law 113) to the Vocational Rehabilitation Service, a readily available mechanism for solving this problem.

Physicians themselves are also partly responsible for the existence of so many unrepaired hernias. The commonest hernia, indirect inguinal in type, represents a congenital defect which often is apparent in infancy or childhood, a time when repair is relatively simple and lastingly effective. Despite this, there seems to be considerable reluctance on the part of some practitioners to recommend infants and children for operation. It is shocking to encounter children wearing trusses year after year. Behavior problems in these children are common and interference with normal growth and development often so severe as to leave permanent psychologic scars in later life. Unfortunately, a few surgeons have unwisely advocated postponement of herniotomy during infancy or early childhood for a number of years, presumably because of lack of confidence in their own technic or because of false sentiment against an operation of choice "at a tender age." Even in adult life, many individuals are not referred for operation by their physicians because of an exaggerated conception of the risk to life or of the probability of recurrence, despite the fact that in the hands of well-trained surgeons and anesthesiologists, the former has been reduced almost to zero, the latter to a few per cent. This is true particularly in older patients, in many of whom the hernia is of long standing and really should have been repaired when they were younger. Nevertheless, operation is all too often not recommended for the aged because of a fear of the risk involved, based largely on statistics published before the time of modern anesthesia, improved surgical technic, and methods of convalescence.

Too many lives are being lost each year because of hernia. Just as important is the fear and disability engendered because the lesion is allowed to remain uncorrected. The hernia problem is a simple one which can be solved by using adequate surgery earlier and more frequently.

ROBERT ELMAN, M.D.

PEPTIC ULCER AND THE ADRENAL STRESS SYNDROME

JOSE Ma. ZUBIRAN, M.D.
ALLAN E. KARK, M.D.
ANTONIO J. MONTALBETTI, M.D.
CLEMENTE J. L. MOREL, M.D.
AND
LESTER R. DRAGSTEDT, M.D., Ph.D.
CHICAGO

THE CONCEPT that duodenal ulcer is in some way due to the stresses and strains of modern life has made much headway in the years since Harvey Cushing focused attention to this problem by his paper on peptic ulcer and the interbrain in 1932.[1] Reference to duodenal ulcer as a psychosomatic disease, or the wound stripe of civilization, gives expression to this point of view. Many physicians have called attention to the high incidence of ulcers in persons whose occupations subject them to continuous mental strain, worry, and anxiety, and to the tendency for exacerbations to occur during periods of emotional stress. How can the tensions of modern life produce disturbances in the central nervous system which affect the stomach and produce a progressive perforating ulcer? One of us [2] has postulated that these events cause a hypertonus in the secretory and motor fibers in the vagus nerves, resulting in excessive continuous secretion of gastric juice and in hypermotility. That such a hypersecretion of gastric juice is the primary cause of duodenal ulcer is indicated by the facts that a hypersecretion of similar extent in experimental animals regularly leads to peptic ulcer formation, and the abolition of this hypersecretion by vagotomy in man causes the duodenal ulcers to heal. Recently, Gray and his associates [3] have suggested an alternate mechanism by means of which chronic stress may play an etiological role in duodenal ulcer. Thus it has been postulated that in response to emotional and systemic stress the cells of the anterior hypothalamus secrete a humoral substance which stimulates the pituitary to release corticotropin. Corticotropin then increases the liberation of adrenal corticoids, such as cortisone, from the adrenal cortex, and these in turn stimulate the gastric glands to an increased production of pepsin and hydrochloric acid. This

From the Department of Surgery, University of Chicago, The School of Medicine.

The supplies of cortisone used in this research were generously provided by Dr. Lawrence L. Lachat, of The Armour Laboratories.

This work has been aided by grants from the Otho S. A. Sprague Memorial Institute, and from the Division of Research Grants and Fellowships of the National Institutes of Health, United States Public Health Service.

1. Cushing, H.; Surg., Gynec. & Obst. **55**:1, 1932.

2. Dragstedt, L. R.: Surg., Gynec. & Obst. **83**:547, 1946.

3. Gray, S. J.; Benson, J. A. Jr.; Reifenstein, R. W., and Spiro, H. M.: J. A. M. A. **147**:1529, 1951.

theory accounts for the hypersecretion of gastric juice, in response to stress, by a mechanism independent of the vagus-nerve supply to the stomach, and if valid would remove an important consideration for the employment of vagotomy in the treatment of duodenal ulcer.

When corticotropin and cortisone were first used as therapeutic agents, there were conflicting reports in the literature of the effect of these drugs on peptic ulcers. Some investigators believed that corticotropin and cortisone had a beneficial effect on these lesions.[4] Many recent observations have indicated, however, that both these agents may produce an exacerbation of ulcer symptoms even leading to perforation.[5] Both Gray and his associates, and Zubiran, Kark, and Dragstedt [5c] have reported that the administration of corticotropin produces a sustained and marked increase in the secretion of gastric juice.

The following experiments were performed in an attempt to find out the effect and mode of action of cortisone on gastric secretion in experimental animals.

EXPERIMENTAL PROCEDURE

The observations to be described were carried out on dogs provided with a Pavlov or a Heidenhain pouch and on animals with total stomach pouches. In two of these animals the vagus innervation of the isolated pouch was preserved [5d] (Dragstedt pouch), and in two the vagus supply to the pouch was divided [5e] (Lim-Ivy pouch). After these animals recovered from the operation, they were fed a standard diet consisting of horse meat, a proprietary dog food, and milk. All the animals remained in excellent health and for the most part maintained the preoperative body weight. The gastric secretion was collected in rubber football bladders attached to the nylon plastic cannulae, and these were emptied once in each 24-hour period. The volume was measured and the juice titrated for free acid using Töpfer's reagent. The 24-hour output of free hydrochloric acid was calculated in milliequivalents by multiplying the volume, expressed in liters, by the free acidity, expressed in clinical units.

These animals had been maintained for a long period of time (9 to 36 months) so that the secretory activity of the isolated stomach or small stomach pouch was well known.

After a control period during which daily collections of gastric juice were made, the dogs were injected subcutaneously with 100 mg. of cortisone once every 24 hours for a period of 14 to 30 days. At the end of administration of the drug the gastric secretion was collected daily for a second control period, which was subdivided into an initial 10-day and a subsequent 20-day period. In two of the animals the experiment was repeated.

RESULTS

The data are summarized in Tables 1 and 2.

Dog 426: A Heidenhain Pouch.—During the preliminary control period 21 quantitative 24-hour collections of gastric juice were made, which revealed an average daily volume of 375 cc., with a free-acid concentration of 133 clinical units and a calculated free-acid output of 50 mEq. per day. Cortisone was then administered daily for 16 days, and during this time the average daily volume increased to 492 cc., with a free-acid concentration of 145 clinical units and a calculated free-acid output

4. Sandweiss, D. J.; Saltzstein, H. C.; Scheinberg, S. R., and Parks, A.: J. A. M. A. **144**:1436, 1950.

5. (*a*) Kirsner, J. B.: Klotz, A. P., and Palmer, W. L.: Gastroenterology **20**:27, 1952. (*b*) Beck, J. C.; Browne, J. S. C.; Johnson, L. G.; Kennedy, B. J., and Mackenzie, D. W.: Canad. M. A. J. **62**:423, 1950. (*c*) Zubiran, J. M.; Kark, A. E., and Dragstedt, L. R.: To be published. (*d*) Dragstedt, L. R., and Ellis, J. C.: Am. J. Physiol. **93**:407, 1930. (*e*) Lim, R. K. S.; Ivy, A. C., and McCarthy, J. E.: Quart. J. Exper. Physiol. **15**:13, 1925. (*f*) Sandweiss.[4]

TABLE 1.—*Effect of Cortisone on Gastric Secretion*

Preparation	Control				Cortisone				Control				Control			
	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl
Dog 426, Heidenhain pouch	21	375	133	50 ± 2.4	16	492	145	71 ± 5.0	20	465	124	58 ± 4.3	..	...	...	.....
Dog 307, Heidenhain pouch without antrum	30	41	28	1.1 ± 0.18	25	53	65	3.4 ± 0.35	10	54	56	3.2 ± 0.45	20	35	20	0.7 ± 0.13
Dog 382, Pavlov pouch	30	513	138	71 ± 3.6	18	623	148	92 ± 7.8	10	610	144	88 ± 5.9	20	316	132	42 ± 3.3
Dog 425, total pouch with vagus nerves	20	341	113	38 ± 2.2	14	481	126	61 ± 5.1	10	481	129	62 ± 3.4	20	382	105	40 ± 1.9
Dog 425, experiment repeated	24	337	108	36 ± 2.5	28	398	123	49 ± 3.1	10	361	116	43 ± 4.4	20	333	97	32 ± 1.3

TABLE 2.—*Effect of Cortisone on Gastric Secretion*

Preparation	Control				Cortisone				Control				Control			
	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl	Days	Vol., Cc.	Free Acid	HCl
Dog 189, total pouch with vagus nerves	30	309	71	22 ± 1.1	28	310	96	30 ± 1.4	10	303	70	21 ± 1.4	20	253	65	16 ± 1.4
Dog 417, total pouch without vagi	15	256	112	29 ± 3.5	14	322	123	40 ± 3.2	10	325	114	37 ± 3.9	20	277	106	29 ± 2.5
Dog 417, experiment repeated	30	142	115	16 ± 4.9	21	217	129	28 ± 3.2	10	322	126	40 ± 4.4	20	136	113	15 ± 2.4
Dog 962, total pouch without vagi (dog died)	30	72	54	3.9 ± 0.4	28	81	77	6.2 ± 0.6	..	...	...	.....	..	...	...	.....

of 71 mEq. per day. After cessation of cortisone administration, 20 daily collections of gastric juice were made. The average volume fell to 465 cc., with a free-acid concentration of 124 clinical units, giving an average free-hydrochloric-acid output of 58 mEq. per day. In this experiment the administration of cortisone produced a sustained increase in secretion from a vagus-denervated accessory stomach pouch, the volume increasing by 31%, the free-acid concentration by 9%, and the total free-acid output by 42%.

Dog 307: A Heidenhain Pouch with Antrum of Stomach Extirpated and Intestinal Continuity Reestablished by Gastroduodenostomy.—During a preliminary control period 30 daily collections of gastric juice were made, revealing an average volume of 41 cc., with a free-acid concentration of 28 clinical units and a calculated average free-acid output of 1.1 mEq. per day. Cortisone was then administered daily for 25 days, and during this period the average volume of secretion increased to 53 cc., with an average concentration of free hydrochloric acid of 65 clinical units and an average free-hydrochloric-acid output of 3.4 mEq. per day. The administration of cortisone was then discontinued, and during the first 10 days the average daily volume of gastric secretion was 54 cc., with a free-acid concentration of 56 clinical units and a calculated free-acid output of 3.2 mEq. per day. During the following 20 days the average volume of secretion fell to 35 cc., with a free-acid concentration of 20 clinical units and a calculated free-acid output of 0.7 mEq. per day. In this animal, which had had a resection of the gastric antrum and had no vagus innervation to the isolated pouch, the output of gastric juice under the influence of cortisone increased by 29%, the free acidity by 132%, and the free hydrochloric acid by 209%. It should be noted, however, that in a preparation of this type, in which both the nervous and the antrum phases of secretion are absent, the volume of secretion is so meager that the relative response to a stimulating agent such as cortisone may be very marked. It is also evident that the stimulating effect of cortisone persisted for approximately 10 days after the drug was discontinued.

Dog 382: A Pavlov Pouch.—During the preliminary control period 30 daily collections of gastric juice were made from the pouch, revealing an average daily volume of 513 cc., with a free-acid concentration of 138 clinical units and a calculated free-acid output of 71 mEq. per day. During the 18-day period of administration of cortisone the average daily gastric secretion increased to 623 cc. per day, with a free-acid concentration of 148 clinical units and a calculated free-acid output of 92 mEq. The administration of cortisone was then discontinued, and during the first 10 days the average secretion of gastric juice persisted at 610 cc., with a free-acid concentration of 144 clinical units and a calculated free-acid output of 88.5 mEq. per day. During the next 20 days the average daily gastric secretion fell to 316 cc., with a free-acid concentration of 132 clinical units and a calculated free-acid output of 42 mEq. per day. In this animal, with a functioning antrum and with intact vagus innervation of the accessory stomach pouch, the administration of cortisone increased the volume of gastric secretion by 17%, the free-acid concentration by 7.2%, and the total free-acid output by 29.5%. It is again evident that the stimulating effect of cortisone persisted for about 10 days after the drug was discontinued.

Dog 425: A Total Stomach Pouch with Vagus Innervation Preserved (Dragstedt Pouch).—During the preliminary control period 20 daily collections of gastric juice were made, revealing an average volume of 341 cc., with an average free-hydro-

chloric-acid concentration of 113 clinical units and a calculated free-acid output of 38 mEq. per day. Cortisone was then administered daily for a period of 14 days, and during this time the average daily volume increased to 481 cc., with a free-acid concentration of 126 clinical units and a calculated free-acid output of 61 mEq. per day. After cessation of cortisone administration, during the first 10 days the volume of secretion persisted at 481 cc. per day, with a free-acid concentration of 129 clinical units and a calculated free-acid output of 62 mEq. per day. During the next 20 days, however, the average volume decreased to 382 cc., with a free-acid concentration of 105 clinical units and a calculated free-hydrochloric-acid output of 40 mEq. per day. This experiment was repeated two months later in the same animal and with the following results. During the preliminary control period 24 daily collections of gastric juice were made, revealing an average daily volume of 337 cc., with a free-acid concentration of 108 clinical units and a calculated free-acid output of 36 mEq. per day. Cortisone was then administered for a period of 28 days, and during this time the average volume of secretion increased to 398 cc., with a free-acid concentration of 123 clinical units and a calculated free-acid output of 49 mEq. per day. Cortisone administration was then discontinued, and for the first 10 days the average volume continued at 361 cc., with an average free-acid concentration of 116 clinical units and a calculated free-acid output of 43 mEq. per day. During the next 20 days the average daily volume decreased to 333 cc., with a free-acid concentration of 97 clinical units and a calculated total-acid output of 32 mEq. per day, or approximately the same as during the preliminary control period. In this animal, with antrum intact but not in contact with food, and with vagus innervation to the pouch preserved, the output of hydrochloric acid in response to the administration of cortisone was increased by 60% during the first experiment and by 36% in the second experiment.

Dog 189: A Total Stomach Pouch with Vagus Innervation Preserved but with Antrum Removed.—During the preliminary control period 30 24-hour collections of gastric juice were made, revealing an average daily volume of 309 cc., with an average free-acid concentration of 71 clinical units and a calculated average free-hydrochloric-acid output of 22 mEq. per day. Cortisone was then administered daily for a period of 28 days, and during this time the average volume of gastric secretion was 310 cc. per day, with a free-acid concentration of 96 clinical units and a calculated free-acid output of 30 mEq. per day. After cessation of cortisone administration, during the first 10 days the volume of secretion persisted at an average of 303 cc. per day, with a free-acid concentration of 70 clinical units and a calculated free-acid output of 21 mEq. per day. During the next 20 days the average daily volume of secretion fell to 253 cc., with a free-acid concentration of 65 clinical units and a calculated free-acid output of 16 mEq. per day. In this animal, with a total gastric pouch with intact vagus innervation but with the antrum extirpated, the administration of cortisone produced an increase in the output of free hydrochloric acid of 36% above the control level.

Dog 472: A Total Gastric Pouch Without Vagus Innervation (Lim-Ivy Pouch).—During the preliminary control period 15 daily collections of gastric juice were made, revealing an average volume of 256 cc., with a free-acid concentration of 112 clinical units and a calculated daily output of 29 mEq. per day. Cortisone was then administered daily for 14 days, and during this time the average volume of gastric

secretion increased to 322 cc., with a free-acid concentration of 123 clinical units and a calculated total free-hydrochloric-acid output of 40 mEq., or an increase of 38% above the control level. During the first 10 days following cessation of cortisone administration, the average daily volume of secretion persisted at 325 cc., with a free-acid concentration of 114 clinical units and a calculated free-hydrochloric-acid output of 37 mEq. During the next 20 days, however, the average volume fell to 277 cc., with a free-acid concentration of 106 clinical units and a calculated free-acid output of 29 mEq. The experiment was repeated again in this same animal after a rest period. During the preliminary control period 30 daily collections of gastric juice were made, revealing an average output of 142 cc. per day, with a free-acid concentration of 115 clinical units and a calculated free-hydrochloric-acid output of 16 mEq. per day. Cortisone was then administered for a period of 21 days, and during this time the average volume of gastric juice increased to 217 cc. per day, with a free-acid concentration of 129 clinical units and an average calculated free-hydrochloric-acid output of 28 mEq. per day, or an increase of 75% above the control period. During the first 10 days after the cessation of cortisone administration, the average daily volume of secretion persisted at 322 cc., with a free-acid concentration of 126 clinical units and a calculated free-hydrochloric-acid output of 40 mEq. per day. During the next 20 days the average daily volume of secretion decreased to 136 cc., with a free-acid concentration of 113 clinical units and a calculated free-hydrochloric-acid output of 15 mEq.

Dog 962: A Total Vagus-Denervated Gastric Pouch with Antrum Resected.—During the preliminary control period 30 daily collections of gastric secretion were made, revealing an average volume of 72 cc. per day, with a free-acid concentration of 54 clinical units and a calculated free-hydrochloric-acid output of 3.9 mEq. per day. Cortisone was then administered for 28 days, and during this period the average daily volume of secretion was 81 cc., with a free-acid concentration of 77 clinical units and a calculated free-hydrochloric-acid output of 6.2 mEq., or an increase of 59% above the control level.

COMMENT

The daily subcutaneous administration of 100 mg. of cortisone for a period of 14 to 30 days produced a sustained increase in the output of acid in the gastric secretion of dogs, ranging from 30 to 75% above the preliminary control level. This hypersecretion induced by cortisone persisted for 7 to 10 days after administration of the drug was discontinued. It has been amply demonstrated that a hypersecretion of gastric juice is the major factor in the production of duodenal ulcers, whether this hypersecretion is produced by continuous stimulation with histamine,[6] by hyperactivity of the antrum mechanism,[7] or by hyperactivity of the nervous mechanism of secretion.[8] The increase in gastric secretion observed in these experiments could thus account for the onset of ulceration or aggravation of clinical symptoms sometimes seen during the administration of cortisone. The increase in gastric secretion occurred in animals with isolated pouches to which the vagus innervation had been removed as well as in those in which the nerves were intact. It also occurred in

6. Hay, L. J.; Varco, R. L.; Code, C. F., and Wangensteen, O. H.: Surg., Gynec. & Obst. **75**:170, 1942.

7. Dragstedt, L. R.; Oberhelman, H. A., Jr., and Smith, C. A.: Ann. Surg. **134**:332, 1951.

8. Dragstedt, L. R.; Woodward, E. R.; Neal, W. B., Jr.; Harper, P. V., Jr., and Storer, E. H.: Arch. Surg. **60**:1, 1950.

animals in which the gastric antrum was present and in those in which it had been extirpated. It is thus probable that cortisone acts directly on the gastric glands or on the peripheral neuroglandular mechanism and not on the vagus nerves or through the release of gastrin from the antrum mucosa.

In a large series of duodenal-ulcer patients studied in this clinic in whom a profound hypersecretion of gastric juice in the fasting stomach was found before operation, this hypersecretion was reduced to approximately the normal level in 98% of patients by a complete gastric vagotomy. If the hypersecretion in these patients were due to the adrenal stress mechanism postulated by Gray and his associates, we should expect from the data derived from this study that this hypersecretion would continue after vagotomy, since the stimulating effect of cortisone on the gastric glands persists even after both vagus denervation and extirpation of the antrum. The experiment appears to be a conclusive one and suggests that in duodenal-ulcer patients the central nervous system plays its undoubted role through increasing the secretion of gastric juice by excessive impulses over the vagus nerves and not through the release of corticotropin with its secondary effect of augmenting the output of cortisone from the adrenal glands.

SUMMARY

The subcutaneous administration of 100 mg. of cortisone daily for periods of 14 to 30 days produced a sustained increase in gastric secretion in dogs, which persisted for 7 to 10 days after administration of the drug was discontinued. This stimulating action was observed in dogs provided with stomach pouches free from vagus innervation as well as in those in which the vagus nerve supply was intact. It was found also in animals with isolated stomach pouches when the antrum had been extirpated. It is thus evident that the stimulating action of cortisone is exerted directly on the gastric glands. Since gastric vagotomy abolishes the hypersecretion of gastric juice in the fasting stomach of duodenal-ulcer patients, it is evident that this hypersecretion is produced by excessive secretory impulses over the vagus nerves and is not due to the liberation of cortisone from the adrenal glands as a part of the so-called stress phenomena.

SURGICAL TREATMENT OF COMPLETE RECTAL PROLAPSE

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THE MULTIPLICITY of operative procedures advocated for the treatment of complete rectal prolapse is indicative of the difficulties encountered in effecting relief of this condition. These procedures may be classified as suspensions, resections, and hernioplasties, or various combinations of these. If complete rectal prolapse is considered to be a hernia, hernioplasty directed toward repair of the musculofascial defects is the most logical approach to the problem. Our experience in treating 20 patients with complete rectal prolapse indicates that an abdominal-perineal plastic repair, as carried out in 6 of the patients, is an effective treatment.

Certain anatomical weaknesses are present when severe complete rectal prolapse occurs. The rectosigmoid colon is of unusual length. The rectosigmoid mesentery is long. The "rectal stalks" containing middle hemorrhoidal vessels are elongated. The external anal sphincter is dilated. The levatores ani are separated, the anterior and posterior rectal walls are mobile and the cul-de-sac is deep. When the prolapse is primarily of the anterior rectal wall and the prolapse curves posteriorly upon extrusion, it may be considered a sliding hernia of the cul-de-sac. This observation

Most cases of complete rectal prolapse represent sliding hernias of the cul-de-sac. In these patients the outstanding defects are a deep cul-de-sac, a mobile anterior rectal wall, a separation of the levatores ani, and a laxity of the external anal was emphasized by Moschcowitz.[1]
sphincter. The correction of these defects by an abdominal-perineal approach has given encouraging results.

The probability of a poor result is increased greatly by mental deficiency, insanity, or a deficient anal sphincter tone due to neurologic disorder. The frequency of rectal prolapse in patients with such disorders (4 of the 20 patients in this series) may be related to neural defects with associated poor muscular support of the rectum. These four patients were or had been inmates of "mental institutions," which provided an unusually bulky (high-residue) diet. It is postulated that such a diet might predispose patients to rectal prolapse. One recurrence of complete rectal prolapse after abdominal-perineal hernioplasty was in a patient who had neurosyphilis, psychosis, and unusually poor external anal sphincter tone.

From the Department of Surgery, State University of Iowa College of Medicine.

1. Moschcowitz, A. V.: The Pathogenesis, Anatomy, and Cure of Prolapse of the Rectum, Surg., Gynec. & Obst. **15**:7-21, 1912.

In this series of 20 patients there were 13 females and 7 males. The age range was from 16 to 83 years. The condition occurred most frequently in those patients in the fourth, fifth, or sixth decade of life.

TECHNIQUE OF OPERATION

Abdominal-perineal plastic repair of complete rectal prolapse is carried out in the following manner. The abdomen is entered through a lower abdominal incision. The pelvic peritoneum is

Fig. 1.—Abdominal approximation of levatores ani.

opened, and the rectum is mobilized and drawn cephalad, assuring reduction of the prolapse. Mobilization of the rectosigmoid colon in the hollow of the sacrum is followed by scarring with probable fixation of the posterior rectal wall, as described by Pemberton.[2] The "rectal stalks"

2. Pemberton, J. deJ., and Stalker, L. K.: Surgical Treatment of Complete Rectal Prolapse, Ann. Surg. **109**:799-808 (May) 1939.

are not disturbed nor is the sigmoid mesentery shortened. The peritoneal cul-de-sac is excised, and the peritoneum is later repaired at a high level. The levatores ani are approximated in front of the rectum (Fig. 1), and the rectal sheath is sutured to the sheaths of structures anterior to the rectum. Rectal, vaginal, prostatic, and bladder sheaths represent condensations of the visceral layer of pelvic fascia around these organs.[3] This procedure provides fixation of the anterior rectal wall and obliteration of the space left by removal of the deep peritoneal cul-de-sac. This abdominal phase of the repair is essentially that described by Graham.[4]

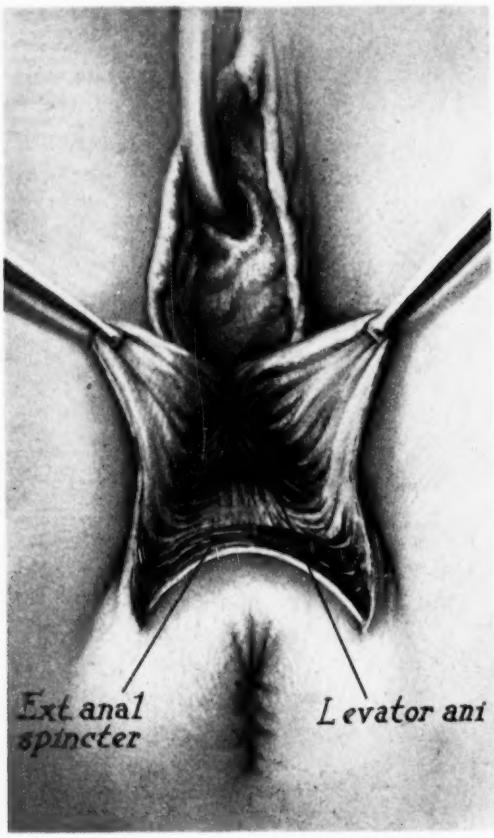

Fig. 2.—Perineal approximation of levatores ani.

After the abdominal incision is closed, the perineal portion of the operation is done. In the female the perineal procedure consists of posterior vaginal repair. By this method additional approximation of levatores ani is accomplished (Figs. 2 and 3), and fixation of rectal and vaginal

3. McGregor, A. L.: A Synopsis of Surgical Anatomy, Ed. 5, Baltimore, Williams & Wilkins Company, 1943.

4. Graham, R. R.: The Operative Repair of Massive Rectal Prolapse, Ann. Surg. **115**:1007-1014 (June) 1942.

sheaths is reinforced by additional sutures. The external anal sphincter is reduced in size by plication sutures if it is very lax. In the male a transverse semilunar incision is made midway between the rectum and scrotum. The space between rectal sheath and prostatic sheath is then developed. The levators are approximated, rectal and prostatic sheaths are sutured, and the external anal sphincter is narrowed by plication.

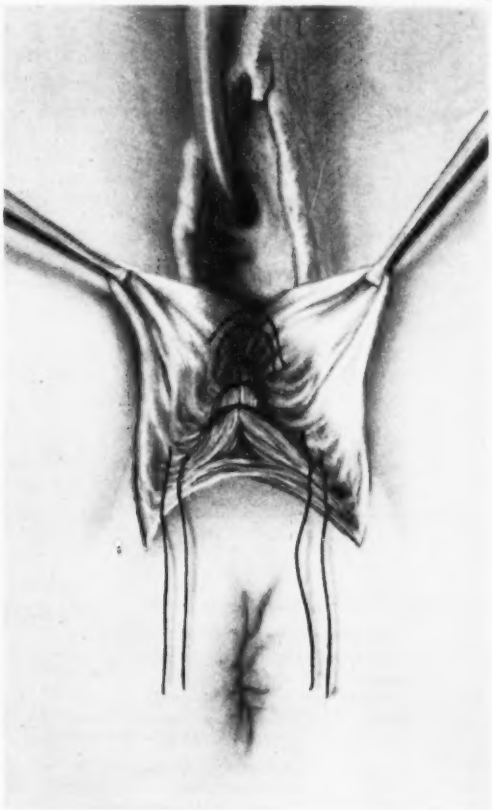

Fig. 3.—Perineal approximation of levatores ani and perineal body reconstruction.

RESULTS

The cul-de-sac obliteration procedure, as described by Moschcowitz, was carried out in 12 patients. There were no operative deaths. Follow-up information was obtained in 11 patients. Eight of these had recurrence of the prolapse. The repair remained stable in three patients. Table 1 summarizes the results obtained by the Moschcowitz operation.

Sigmoidopexy was employed in one patient and was followed by incomplete (mucosal) prolapse.

Transabdominal sigmoid resections were carried out in four patients. One patient in whom resection was carried out as the primary procedure had relief of her prolapse until her death from pneumonia one year later. Two of these patients had had recurrence of the prolapse following repair by the Moschcowitz procedure. Of these two patients one died 10 days postoperatively in a shock-like state thought

TABLE 1.—*Results of Surgical Treatment of Complete Rectal Prolapse, Moschcowitz and Miscellaneous Procedures (1940 to 1950)*

Case	Age, Yr.	Sex	Date of Operation	Procedure	Result
E. M.	46	M	5/20/40	Moschcowitz	Recurrence (1 yr.)
M. S.	56	F	10/17/40	Moschcowitz	Good result (6 yr.)
D. B.	42	F	6/ 4/41 9/10/42	Moschcowitz, levator repair	Recurrence (3 mo.) Recurrence †
R. H.	57	F	9/12/41	Moschcowitz	No follow-up
E. C.*	50	F	12/ 8/41	Moschcowitz	Good result (5 yr.)
L. D.	61	M	4/ 6/43	Moschcowitz	Recurrence †
M. D.	46	F	4/16/43	Moschcowitz	Recurrence †
A. G.*	16	F	5/12/43 10/ 9/46	Moschcowitz, sigmoid resection	Recurrence † Recurrence (18 mo.)
W. L.	46	M	8/ 3/43	Sigmoidopexy	Recurrence (3 yr.)
V. V.*	24	M	10/13/43 5/18/44	Moschcowitz, Anterior resection sigmoid	Recurrence (1 mo.) Died (10 days after operation)
E. M.	71	F	12/30/43	Moschcowitz	Good result (2½ yr.)
M. H.	83	F	8/24/44	Mickulicz resection of sigmoid	Good result (1 yr.)
E. H.	63	F	3/ 6/46	Moschcowitz	Recurrence †
E. Mc.	24	F	3/19/49 12/18/51	Moschcowitz, sigmoidopexy	Recurrence (9 mo.); good early postoperative result

* Demented or mentally defective patient.
† The exact interval between operation and recurrence unknown.

TABLE 2.—*Results of Surgical Treatment of Complete Rectal Prolapse, Abdominal-Perineal Plastic Repair (1948 to 1950)*

Case	Age, Yr.	Sex	Date of Operation	Procedure	Result
C. C.	83	M	11/10/48	Abdominal-perineal hernioplasty	Good result (3 yr.)
D. G.	25	M	6/13/49	Abdominal-perineal hernioplasty	Recurrence (Slight) (3 mo.)
H. M.	65	F	8/ 5/49	Abdominal-perineal hernioplasty	Good result (2 yr.)
A. D.	68	M	9/16/49	Abdominal-perineal hernioplasty	Good result (2 yr.)
V. H.	39	F	4/17/50	Abdominal-perineal hernioplasty	Good result (20 mo.)
E. E.*	44	F	4/27/50 1/17/52	Abdominal-perineal hernioplasty, resection, and "pull-through"	Recurrence (16 mo.); good early postoperative result

* Demented or mentally defective patient.

to be due to peritonitis. Autopsy was not performed. The other patient was treated by obstructive resection of the sigmoid colon and by subsequent colostomy closure leaving the sigmoid colon fixed to the abdominal wall. Recurrence occurred 18 months later. In the fourth patient rectosigmoid resection combined with a "perineal pull-through" procedure was carried out recently for recurrence following abdominal-perineal repair of a "cul-de-sac sliding hernia." Insufficient time has elapsed since this procedure to evaluate the result. Tables 1 and 2 indicate the results obtained by resection of the colon.

Only one patient was treated by a transabdominal approximation of the levatores ani. Recurrence of the prolapse ensued.

During the past three years, six patients with complete rectal prolapse have been treated by the abdominal-perineal repair described above (Table 2). Of the six patients treated by abdominal-perineal repair, four have had no recurrence 20 to 36 months after operation. In one (E. E.) complete recurrence developed 16 months after the initial operation. Another patient (D. G.) has not been reexamined, but a letter from him states that a slight prolapse has developed with defecation three months postoperatively. The patient with complete recurrence (E. E.) is demented as a result of neurosyphilis and has poor anal sphincter tone. This recurrence was treated recently by abdominal-perineal resection of the rectosigmoid colon and "perineal pull-through."

SUMMARY

Six patients with complete rectal prolapse of the anterior rectal wall ("sliding hernia") have been treated by an abdominal-perineal plastic repair.

Correction of the prolapse was obtained in four patients. One patient was improved but not "cured." Complete recurrence occurred in another patient.

The results obtained by the use of this method are so encouraging that it is recommended as the preferable method of primary surgical treatment. The more radical procedure of resection of the prolapsed bowel is justified as a secondary procedure in cases of recurrence.

Prognosis should be guarded in mentally defective and insane patients and in those in whom there is a primary neurologic defect resulting in poor anal sphincter tone.

PARACARINAL BIOPSY IN EVALUATION OF OPERABILITY OF CARCINOMA OF THE LUNG

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IN EXAMINING a patient with carcinoma of the lung, it is the commonly accepted practice to search for evidence of metastases. If this is found, the condition with rare exceptions is rightly considered inoperable from the standpoint of cure of the disease. An additional factor to be considered is the location of the growth with respect to the trachea and the carina. If either of these structures is involved by the growth, the ordinary type of pneumonectomy could hardly be expected to be sufficient for cure. The neoplasm or its extension may be so situated as to render the growth obviously inoperable. Not infrequently, however, a growth visualized at a considerable distance from the carina extends in the submucosal lymphatics to the trachea and even to the opposite bronchus without producing any gross changes. In the absence of any other evidence of metastasis, such a lesion would ordinarily be considered to be suitable for pneumonectomy even after thorough clinical, roentgenological, and bronchoscopic examination. Recognition of such proximal extension of the growth is the aim of paracarinal biopsy.

The tendency of bronchial carcinoma in some cases to spread proximally in this way without any visible evidence has been known for many years. At an autopsy performed by one of us in 1927 on a patient with a small-cell carcinoma originating in the terminal portion of the right lower lobe bronchus, we were able to demonstrate nests of carcinoma cells in the submucosal lymphatics of the upper thoracic part of the trachea in spite of the fact that the tracheobronchial tree appeared normal grossly except at the site of the original tumor. Since then the observation that proximal extension of the lesion not infrequently takes place in this way has been stressed on several occasions.[1]

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Read before the Section on Diseases of the Chest at the One Hundred First Annual Session of the American Medical Association, Chicago, June 10, 1952.

The bronchoscopic examinations and paracarinal biopsies were performed by all members of the staff of the Otolaryngological Department of the Mount Sinai Hospital.

1. Rabin, C. B.: Relation of Roentgenography of the Chest to the Laryngologist: Atelectasis and Neoplasms of the Lungs, Laryngoscope **42**:923, 1932. Rabin, C. B., and Neuhof, H.: A Topographic Classification of Primary Cancer of the Lung: Its Application to the Operative Indication and Treatment, J. Thoracic Surg. **4**:147, 1934.

In 1944, we deliberately excised a portion of grossly normal mucous membrane of the right main bronchus in a patient with carcinoma of the right lower lobe because the biopsy specimen of the original tumor in the lower lobe showed carcinoma cells in the lymphatics. Nests of carcinoma cells were also found in the submucosal lymphatics of the right main bronchus, and therefore the condition was considered inoperable. Obvious lymphatic metastases developed a short time thereafter.

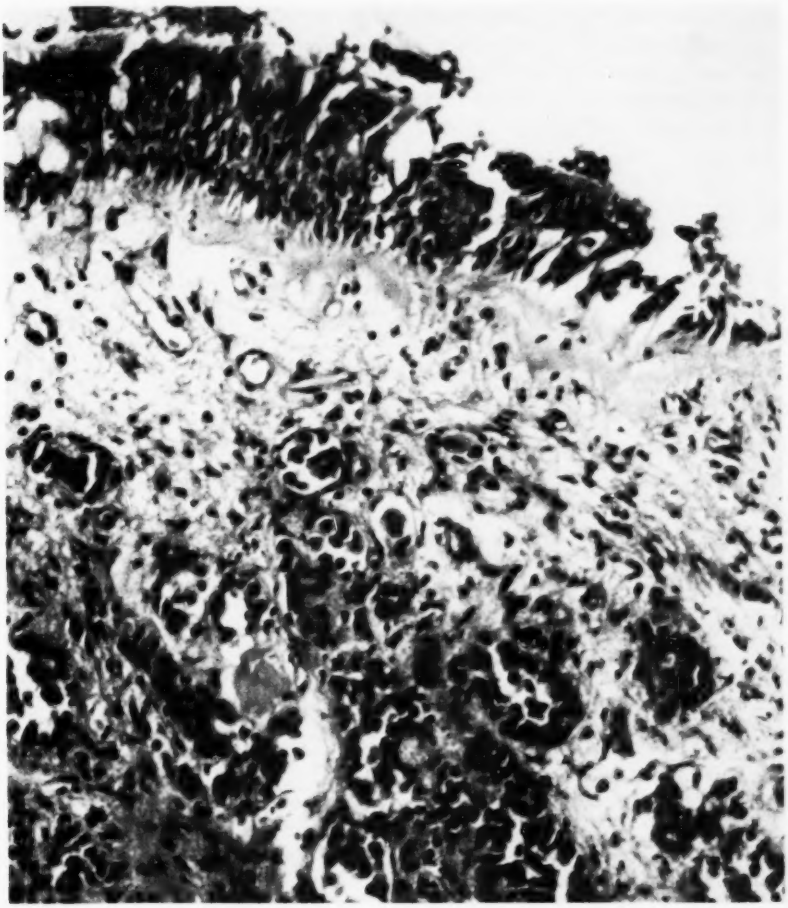

Fig. 1.—Biopsy specimen of bronchial mucous membrane taken near the carina, showing normal surface epithelium and carcinoma cells in the submucosal lymphatics. The primary tumor was in the bronchus of the right lower lobe.

The frequency of local proximal extension of carcinoma of the bronchus invisible to the bronchoscopist was reported by Griess, McDonald, and Clagett in 1945.[2]

2. Griess, D. F.; McDonald, J. R., and Clagett, O. T.: The Proximal Extension of Carcinoma of the Lung in the Bronchial Wall, J. Thoracic Surg. **14**:362, 1945.

They concluded that the resection of the neoplasm should be carried out at least 1 to 1.5 cm. proximal to all bronchial carcinomas because of the frequency of submucosal extension of the growth.

In 1946 we began a systematic study of the mucous membrane lateral to the carina to determine the frequency of microscopic involvement in this region of the mucosa regardless of whether it appeared to be diseased on gross bronchoscopic examination. The finding of submucosal extension of the carcinoma to the paracarinal region from a more distant bronchial carcinoma was considered an indication of inoperability from the standpoint of cure. Biopsies of tissue from the paracarinal region were positive in a sufficient number of cases to make us conclude that paracarinal biopsy should be performed routinely in the evaluation of the operability of bronchial carcinoma (Fig. 1).

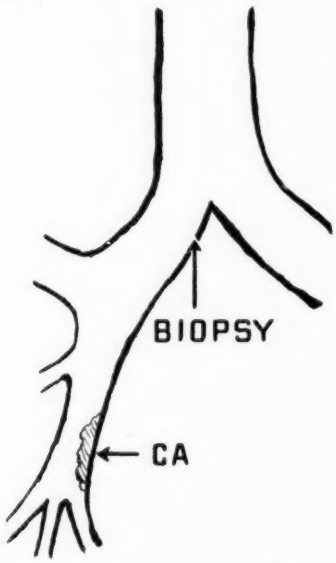

Fig. 2.—Site of biopsy along the side of the carina. The primary carcinoma was in the bronchus of the right lower lobe at a distance from this site.

MATERIAL AND METHOD

In the period between June, 1946, and October, 1951, a paracarinal biopsy was performed in 154 cases of primary carcinoma of the lung. In 141 of these the diagnosis of carcinoma was confirmed by tissue examination either at bronchoscopy, exploratory operation, or examination of a metastatic lesion. In 13 of the cases there was no microscopic proof of the presence of carcinoma but the clinical course was sufficiently characteristic for a positive diagnosis. The biopsy specimen was taken within 1 cm. of the carina on the side of the primary carcinoma whether or not there was evidence of gross disease in this region (Fig. 2). An attempt was always made to make the bronchoscopic punch deep enough to obtain submucosal tissue. A biopsy specimen was also taken at the region where the primary carcinoma was suspected. This was done even when the bronchi presented no gross abnor-

mality; the tissue for biopsy was then taken in the region suggested by the location of the lesion as determined from the roentgenological examination. Since tissue might unknowingly be carried in the bronchoscopic forceps, care was taken either to use a fresh forceps or to clean the original forceps thoroughly before using it for the paracarinal biopsy.

INCIDENCE OF POSITIVE PARACARINAL BIOPSY

Of the 154 cases studied, biopsy of tissue obtained from the paracarinal region was positive in 31, or approximately 20%. In 16 of these, the mucous membrane at the site of biopsy appeared perfectly normal on gross bronchoscopic examination, yet microscopic examination of the excised tissue showed the presence of carcinoma, usually in the submucosal lymphatics (Table 1).

In 30 other cases gross changes of greater or less degree were visible, but in only half of these could carcinoma be detected on microscopic examination. The

TABLE 1.—*Incidence of Positive Paracarinal Biopsy*

	Cases, No.	Cases, %
Total	154	....
Paracarinal biopsy positive	31	20.1
Gross involvement	15	48.4
No gross changes on bronchoscopy	16	51.6

TABLE 2.—*Evaluation of Operability* Before Paracarinal Biopsy: 154 Cases*

Inoperable†		Operable	
Metastases	39	Before paracarinal biopsy	106†
Gross involvement of trachea or carina, proved microscopically‡	9	Positive paracarinal biopsy with negative bronchoscopic appearance	15
Total	48	Positive paracarinal biopsy with questionable change near carina‡	2

* By operability is meant the absence of known neoplastic involvement by metastases or invasion beyond the line of resection at an ordinary pneumonectomy.

† In 17 cases of 106—16%—paracarinal biopsy was the sole evidence of inoperability.

‡ In 15 cases, gross appearance suggested abnormality at the carina but biopsy was negative.

discrepancy between the gross and microscopic findings in these 30 cases is due partly to the fact that the slightest aberration from normal was considered a gross change. In some, the deviation was undoubtedly within normal limits. In others, the changes noted were due to carcinomatous involvement so deeply situated that the diseased area could not be reached by the bronchoscopic forceps.

RELATION OF THE PARACARINAL BIOPSY TO OPERABILITY

It is significant that in 15 of the 16 patients with a positive paracarinal biopsy despite the absence of gross changes in the mucous membrane at the biopsy site, there was no other evidence of extension of the tumor or of metastasis. The lesions of these patients, therefore, were considered to be operable from the standpoint of cure before the biopsy was done. The lesions of two additional patients with questionable gross changes near the carina, but with microscopic evidence of carcinomatous involvement in that region, also appeared operable. In the entire series of 154 patients, the condition of 48 was found to be inoperable for reasons aside

from the results of paracarinal biopsy. In 39 of these definite evidences of metastases were found before operation, and in the other 9, who had no evidence of metastases, there was found gross evidence of involvement of the trachea or of the carina, proved by microscopic examination to be due to carcinoma. There remain 106 patients whose lesions were considered to be operable before the results of microscopic examination of tissue removed from the paracarinal region was available. For these, the paracarinal biopsy provided the sole contraindication to operation in 17 (Table 2). This incidence of 17 positive paracarinal biopsies for 106 patients whose condition otherwise appeared operable, approximately 16% of this series, is a significant one.

CORRELATION OF FINDINGS OF PARACARINAL BIOPSY WITH OTHER OBSERVATIONS

The material was analyzed from the standpoint of the location of the primary bronchial tumor, its gross type as judged by the roentgenological appearance, the gross bronchoscopic appearance of the primary growth, the microscopic morphology, the duration of the symptoms, and the age and sex of the patient. As a result of this analysis it appeared that the incidence of positive paracarinal biopsies of grossly

TABLE 3.—*Correlation of Results of Paracarinal Biopsy with Duration of Symptoms*

Duration of Symptoms, Mo.	No.	Positive, Paracarinal Biopsy	
		Total	Grossly Negative
0-3	79	12	8
3-9	47	13	6
Over 9	28	6	2

normal mucous membrane was not affected by the known duration of the disease. As might be suspected, paracarinal biopsy was positive more frequently when the main bronchus was the site of the primary carcinoma. For patients with carcinoma of the lobar or branch bronchi, paracarinal biopsy was positive much more frequently when the disease involved the right lung. The age and the sex of the patients did not appear to affect the incidence of positive biopsies. However, the number of females and patients in the younger age groups was too small for statistical evaluation. The same observation is true for the microscopic types aside from squamous-cell carcinoma. However, the material appears to show an increased incidence of positive findings in cases of anaplastic or small-cell carcinoma. A significant number of paracarinal biopsies were positive for the patients with rather circumscribed nodular carcinomas, as depicted roentgenologically, as well as for those with the more diffuse and centrally situated types of neoplasm. The type of bronchoscopic change at the site of the primary tumor did not seem to affect the incidence of positive paracarinal biopsy. However, the patients who had no gross evidence of tumor anywhere in the bronchial tree did not have a positive paracarinal biopsy.

Duration of the Disease.—It would seem that the mucous membrane near the carina would be much less frequently involved in the "earlier" cases of carcinoma, i. e., in those in which symptoms were of the shortest duration. This was not true in our series (Table 3). Of 79 patients who were examined before three months

of symptoms, 12 had a positive paracarinal biopsy, and in 8 of these the paracarinal region appeared uninvolved grossly. Of the remaining 75 patients, 19 who had symptoms upward of three months' duration had a positive biopsy, and in 8 of these the region appeared grossly normal. All five patients who had had symptoms for more than 24 months had a negative biopsy. It is of interest that only 4 of the patients in the first group showed gross evidence of paracarinal invasion, while 11 of the patients with symptoms of more than three months showed gross changes. Apparently the type of carcinoma that spreads submucosally to involve the carinal region is apt to do so early in the disease. In the later stages this involvement becomes grossly evident as the neoplasm grows through the lymphatics to involve the epithelial layer.

Site of the Primary Tumor.—As was to be expected, the paracarinal biopsy was positive most frequently when the primary tumor arose in the main bronchus. Thus 10 of 27 patients with main-bronchus carcinoma proved to have a positive paracarinal biopsy. The incidence was equal on the two sides. However, there was a marked difference on the two sides in the case of neoplasms of the lobar or branch bronchi. Thus, there were 17 positive paracarinal biopsies of 79 such neoplasms on the right

TABLE 4.—*Location of Bronchial Neoplasm in 151 Cases of Paracarinal Biopsy**

	Right Lung		Left Lung	
Bronchus Involved	No. of Cases	Paracarinal Biopsy Pos.	No. of Cases	Paracarinal Biopsy Pos.
Main	16	6	11	4
Upper lobe	45	9	27	1
Lower lobe	27	7	18	1
Middle lobe	7	1	..	..
	95	23 (24%)	56	6 (11%)

* Origin at the carina in one case and uncertain in two.

and only 2 positive biopsies of 45 on the left side (Table 4). This difference appears remarkable and possibly has some relation to a difference in the lymphatic drainage on the two sides. The fact that the left main bronchus is longer than the right may also have a bearing on the low incidence of positive paracarinal biopsies when the carcinoma arises from the branches of the left bronchus.

Gross Bronchoscopic Appearance of the Primary Neoplasm.—It is known that some of the most rapidly spreading bronchial carcinomas are manifest bronchoscopically simply as a thickening or granularity of the mucous membrane with obliteration of the cartilaginous markings, accompanied by more or less bronchial stenosis but without the formation of a distinct endobronchial mass. The presence of a fungating or polypoid mass within the bronchus might suggest that these tumors would not spread as actively toward the carina as might growths which were more intramural. However, a positive paracarinal biopsy was obtained in 5 of 46 cases in which the primary tumor, at a distance from the carina, was visualized as an endobronchial mass (Table 5). Positive findings were somewhat more common in cases of the presumably more infiltrating types of tumor manifested only by thickening, stenosis, or granularity of the mucous membranes (19 of 83 cases). The paracarinal biopsy was negative in the 18 cases in which no gross abnormality could be seen anywhere in the bronchial tree. In these the paracarinal biopsy must thus far be considered to be uninformative.

Microscopic Types.—Although the anaplastic carcinomas appeared to involve the carinal region most frequently (7 of 21 cases), the squamous-cell carcinomas also spread to this region with considerable frequency (Table 6). Of 100 instances of squamous-cell carcinoma, the paracarinal biopsy was positive in 19, and in 9 of them the region appeared grossly normal. Despite the fact that this is the least malignant form of bronchial carcinoma, it is evident that the examination is useful in revealing the squamous-cell carcinoma as well as the others. A positive result of paracarinal biopsy was obtained in the cases of mature as well as of immature neoplasms of this type.

Roentgenological Appearance.—Of the 154 neoplasms studied, 44 appeared as nodular masses more or less peripherally located on the roentgen films. The remaining 110 neoplasms appeared roentgenologically as extensive root infiltrations or atelectasis of a lobe or of the entire lung or were characterized by the presence of pleural effusions, large mediastinal masses, or diffuse lymphagitic infiltrations throughout the lungs. These were obviously manifestations of tumors of the root bronchi or of extensively infiltrating tumors, in the presence of which a positive paracarinal biopsy is not surprising. The paracarinal region was involved by carcinoma in 26 of the 110 cases, and all of the 15 cases in which there was gross evidence of paracarinal involvement occurred in this group. We were somewhat surprised, however, to find the paracarinal biopsy to be positive in 5 of the 44 cases of nodular tumor situated at some distance from the lung root. In all five there was bronchoscopic evidence of the primary tumor, but in none were there any gross changes near the carina. Nevertheless, the paracarinal biopsy was positive. The observations in these five cases point to the importance of performing a paracarinal biopsy even in the case of more peripheral, circumscribed tumors when the primary tumor can be visualized bronchoscopically.

TABLE 5.—*Correlation of Paracarinal Biopsy with Bronchoscopic Appearance of Primary Bronchial Tumor*

Bronchoscopic Appearance	No. of Cases	Positive Paracarinal Biopsy
Normal	18	0
Thickening, stenosis, granularity	83	19
Mass present in bronchus, carina normal	46	5
Mass in bronchus, thickened mucosa near carina	1	1
Mass present, invading paracarina region	6	6
	154	31

TABLE 6.—*Relation of Results of Paracarinal Biopsy to Type of Carcinoma*

Microscopic Type	No. of Cases	Paracarinal Biopsy Pos.
Squamous-cell carcinoma	100	19
Adenocarcinoma	13	2
Anaplastic, including small-cell carcinoma	21	7
Cell type undetermined	7	3
	141*	31

* No microscopic material was available in 13 cases.

COMMENT

The term operability as used in this paper refers to the ordinary type of pneumonectomy with a view to the cure of carcinoma. It does not apply to operations performed for the purpose of palliation or to resection of a portion of the trachea. It is probable that some of the patients whose primary neoplasm was situated in the main bronchus and extended grossly to the carina or the tracheal wall might have been cured by an operation more extensive than simple pneumonectomy. It appears to us, however, that cases in which the primary neoplasm is at a distance from the carina and in which the paracarinal region shows microscopic evidence of involvement of the submucosal lymphatics fall into a different category. Whereas a primary tumor of the main bronchus may be fairly well localized to its point of origin and thus permit some sort of resection, the tumor that spreads proximally from a distance via the lymphatic channels is obviously not at all circumscribed. The fact that involvement of the submucosal structures is present where it is invisible to the naked eye makes the limit of its spread unknown. Such tumors have been described as lymphophilic because of their tendency to widespread lymphatic invasion. Our entire experience with them has shown that their involvement is widespread even though clinical evidence of their extension is lacking. In a considerable number of cases this extension became evident, within a short time after positive results of paracarinal biopsy had been obtained, either in the form of enlargement of the lymph nodes of the neck or mediastinum, a carcinomatous lymphangitis involving the opposite lung, or development of a cancerous pleural effusion. In three additional cases an exploratory operation was performed, and in all there was extensive involvement of the mediastinal lymph nodes. It is therefore most unlikely that even resection of the trachea and the opposite main bronchus, together with the carina, would be helpful in such cases.

The incidence of positive paracarinal biopsies in the absence of significant gross changes in 16% of the patients whose lesions otherwise appeared operable indicates the usefulness of the procedure in the screening of patients before the operation. We feel that this form of biopsy should be performed whenever evidence of a bronchial carcinoma is found at bronchoscopy. It is best to take the specimen at the initial bronchoscopy so that the patient need not be subjected to this form of examination a second time.

In addition to the 15 patients with a positive paracarinal biopsy in whom the region appeared involved grossly, there was a similar number in whom the biopsy proved negative despite an abnormal gross appearance. In some of these the growth proved operable and the gross appearance might have been misleading if a biopsy had not been done. It is therefore important not to rely entirely on such changes as thickening of the mucous membrane and rigidity of the bronchus in concluding that the disease has spread beyond the limits of operability. When there are such changes a biopsy should be done on tissue from the paracarinal region as well as from the main tumor.

The incidence of positive paracarinal biopsies was significant for patients whose clinical history was of short duration as well as for patients with a rather circumscribed growth at a distance from the lung root. A consideration of the duration or roentgenologic appearance of the tumor, therefore, should not serve as a deterrent

to the examination. Thus far we have had no complications as a result of the procedure. There has been no hemorrhage, and there has been no interference with healing of the bronchus at the site of resection.

There have been included in this series 18 cases in which the entire bronchial tree appeared grossly normal although the patient undoubtedly had a carcinoma. In all of these the carinal biopsy showed no evidence of the new growth. It seems reasonable, therefore, in cases of suspected carcinoma in which the bronchi appear normal at bronchoscopy to limit the biopsy to that bronchus which leads to the location of the growth as determined roentgenologically. In the unusual cases in which this biopsy proves positive, the tissue for paracarinal biopsy should be taken at a second bronchoscopy. It is proper to exclude these cases in a statistical study to evaluate the usefulness of the paracarinal biopsy. If this is done the incidence of positive paracarinal biopsies becomes significantly greater than the figures we have presented and the value of the procedure becomes more apparent.

SUMMARY

One hundred fifty-four patients with primary carcinoma of the lung were subjected to biopsy of the mucous membrane of the main bronchus within 1 cm. of the carina. In 30 of these some gross abnormality was visible in this region, but for only half of them did the microscopic examination disclose neoplastic cells. In 16 additional patients the mucous membrane in the paracarinal region appeared grossly normal, yet microscopic examination disclosed submucosal lymphatic extension from the tumor, which originated more distally. Of the patients, 106 appeared to be good subjects for cure by pneumonectomy before the results of the paracarinal biopsy were known. Of these, 17, or 16%, showed microscopic evidence of invasion of the paracarinal region by the growth. A positive result from paracarinal biopsy was obtained regardless of the type of abnormality noted by the bronchoscopist, but no biopsy of the paracarinal region was positive when the bronchus leading to the tumor appeared normal. Hidden lymphatic involvement by carcinoma near the carina occurred as frequently in patients with symptoms of less than three months' duration as in those who had had symptoms for a longer period. No complications resulted from the procedure.

CONCLUSIONS

1. A biopsy specimen immediately lateral to the carina should be taken in all cases of bronchoscopically demonstrable primary carcinoma considered for operation.

2. The paracarinal biopsy should be performed at the time of the first bronchoscopy in the case of an abnormality lower in the bronchial tree suspected of being carcinoma. This should be done whether the mucous membrane adjacent to the carina appears normal or not.

3. Resection of the carina and trachea may be feasible in some cases of gross paracarinal involvement by a neoplasm that arises in that location.

4. A positive paracarinal biopsy of mucous membrane that appears normal indicates the presence of submucosal lymphatic spread and this serves as a contraindication to any type of pneumonectomy performed with a view to cure.

SURGICAL PROCEDURE IN TUMORS OF PAROTID GLAND

Preservation of Facial Nerve and Prevention of Postoperative Fistulas

DANKO RIESSNER, M.D.
ZAGREB, YUGOSLAVIA

WHEN OPERATING in the region of the parotid gland, the surgeon encounters three fundamental difficulties: (1) the face is a delicate cosmetic region, (2) the facial nerve is very liable to lesions, and (3) there always exists the possibility of postoperative (post-traumatic) fistulas of the parotid gland. Accordingly, he faces three hazards in this region: (1) scar, (2) palsy of the facial nerve, and (3) stubborn fistulas of the parotid gland.

Furthermore, in the region of the parotid gland the surgeon encounters tumors representing an especially interesting oncological problem, tumors which make the whole clinical procedure exceedingly complicated. Tumors appear here which, to date, have not been systematized by the pathologist. Clinicians, for their part, are making every effort to clarify the differential diagnosis of the tumors in question, particularly because tuberculous lymphadenitis of this region and sialolithiasis, with its complications, are hard to distinguish clinically from true tumors. Helpful as sialography may be in such differentiation, it is far from reliable.

Most tumors of the parotid belong to a class which, despite all controversies among the pathologists, may for practical purposes be given the name "mixed tumor of the parotid gland." They belong to that group of blastomas which lies midway between the malignant and the benign ones, or at least they will become malignant sooner or later. The surgeon is in a precarious situation indeed. While he has to meet the technical problems and dangers as outlined, the character of the tumor makes it urgent that he carry out a radical operative procedure. Every surgeon has experienced this situation, and all are therefore aware that, with both a methodology and systematology for such operations being nonexistent, there is no operative system to guarantee a degree of safety comparable to that in other operative fields. In no manual dealing with surgery—whether it comes from Anglo-Saxon, German, Romanic, or Slav countries—can one find any operative methodology covering this region.[1] Instead, the manuals contain optimistic suggestions as to how to extirpate parotid tumors from their "well-developed" capsules in a manner generally satisfactory and how to perform dissections using

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1. Kirschner, M.: Allegemeire und spezielle chirurgische Operationslehre, Vol. 2, Berlin, Springer-Verlag, 1935. Orr, G. T.: Operations of General Surgery, Philadelphia, W. B. Saunders Company, 1946. Kleinschmidt, O.: Operative Chirurgie, Ed. 2, Berlin, Springer-Verlag, 1943. Bancroft, F. W., and Pilcher, C., editors: Surgical Treatment of the Nervous System: Cranial and Intracranial Surgery, Tumors, Epilepsy and Cranial Nerve Disorders, Spinal Cord, Autonomic Nervous System, Peripheral Nerves, Philadelphia, J. B. Lippincott Company, 1946.

electrodes, which signal in due time—owing to the contractions of the related muscles of the face—any danger of the facial nerves being damaged. This information is far too little for the surgeon.

In addition to the advice given in manuals on surgery, there are numerous articles in the medical literature expressing dissatisfaction with existing methods and describing efforts to create safer ones. This paper represents such an attempt, its aim being to introduce and justify my method of operating for tumor of the parotid gland. The next section of the paper discusses the complex of modern problems pertaining to this region from the surgical, anatomical, pathophysiological, and technical (operative) point of view. Since it was written with a view to justifying my own procedure, I shall first describe this procedure briefly.

I have solved the cosmetic problem of the scar by making a cut around the external ear with a prolongation forward in the direction of the zygoma and behind along the first part of the sternocleidomastoid muscle. This incision widely exposes the operative region, mobilizes the tumor and the gland from the external ear, and gives an easy approach technically to the upper branch of the facial nerve, of which the position is most constant and the thickness easiest to handle. While dissecting, the surgeon can follow this branch through the parenchyma and, if necessary, through the tumor to the other branches of the facial nerve. A lesion to the gland is not to be feared, since by cutting the auriculotemporal nerve—the secretory nerve of the salivary gland—the surgeon has eliminated the possibility of postoperative fistulas.

SURGICAL ANATOMY AND PHYSIOLOGY OF THE PAROTID GLAND

Studying the surgical anatomy in the region of the parotid gland, I found the articles of Sistrunk and of Pierre Duval, who carried out minute preliminary anatomical studies.[2] Sistrunk concluded that it was necessary to dissect the upper branch of the facial nerve, as the tumor was lower in the great majority of cases, a procedure that would enable the surgeon to save that one branch, at least, when operating. Pierre Duval, however, took quite another course. In his belief that the branches of the facial nerve were uncertain as to both their position and their thickness, he preferred to find the trunk of the facial nerve when starting an operation. He suggested chiseling off the mastoid so as to obtain immediate access to it. Great credit goes to the English surgeon Bailey,[3] who in 1941 published his method of operation on the parotid gland and who revived the forgotten but most important papers of the anatomists Grégoire (1912)[4] and McWhorter (1917),[5] both of whom contributed so much to the systematic and topographic anatomy of the parotid gland and the facial nerve.

2. Padovani, P., and Duval, R.: Tumeurs mixtes de la parotide, ablation totale de la glande avec conservation complete du nerf facial, Mém. Acad. chir. **64**:1313, 1938. Leriche, R.: Ablation totale de la parotide avec conservation complete du facial, ibid. **65**:4, 1939. Cogniaux, P.: Parotidectomie avec conservation du facial supérieur: Opération de Duval; Présentation de deux cas, J. chir. et ann. Soc. belge chir. **38-36**:370, 1939.

3. Bailey, H.: Treatment of Tumors of Parotid Gland, with Special Reference to Total Parotidectomy, Brit. J. Surg. **28**:337, 1941.

4. Grégoire, R.: Le nerf facial et la parotide, J. anat. **48**:437, 1912.

5. McWhorter, G. L.: The Relations of the Superficial and Deep Lobes of the Parotid Gland to the Ducts and to the Facial Nerve, Anat. Rec. **12**:149, 1917.

Today it is known, however, that the parotid gland is not a flat cake of the salivary gland. It is not pierced by the trunk or fibers of the facial nerve in as simple a manner as most anatomy textbooks maintain, i. e., from backward below to forward at top.[6] Grégoire established the bilobed structure of the gland and based his thesis on the genesis of the bilobed structure itself. McWhorter found that the two lobes were connected by an isthmus in the middle and that it was the isthmus which, owing to its position, determined the main division of the facial nerve into an upper and a lower branch. Bailey accepted the bilobed structure of the gland as well as the interlobal position of the facial nerve within it as a basis for his method of operation. As the upper lobe represents a higher percentage, owing to its mass, in the formation of tumors, after Bailey the technique of operation was simplified. In his last publication Bailey reported 54 operations performed without the facial nerves being damaged at all.

During our anatomical studies with operative material and cadavers, my associates and I had several experiences unlike those of Bailey. We started using our operative method as early as 1940. According to our findings, the shape of the parotid gland and also the quantity of glandular parenchyma varied considerably. We discovered one fact concerning the form and position of the gland, namely that the gland never went beyond the arch of the zygomatic bone upward but always stopped on the lower edge of the zygomatic arch as a thinned edge of the flat cake. Its development, position, and shape downward and forward were much more variable.

We discovered a marked bilobed structure of the parotid gland in only two cases. In these we also saw the isthmus as a clearly developed formation. In other cases the isthmus was either so wide that we were unable to observe a bilobed structure, or it must have been exceedingly thin. We were unable to establish an interlobal position of the facial nerve and its branches. We could establish, however, that the principal trunk pierced the backward lower part of the superficial lobe of the parotid gland in such a way that the pes anserinus lay in the superficial lobe. In two further cases the cervicofacial branch went just through the tumor, but we could not say whether it belonged to the superficial or to the deep lobe, for we were unable to see a bilobed structure. It seems to us that owing to the expanding growth of the tumor the tissue of the parotid gland flattens, contracts, and changes in a scar-like manner, so that the structure of the surroundings of the glandular parenchyma cannot be well distinguished, this fact being the reason for difficulty in recognizing the bilobed structure. It is our impression that such a structure undoubtedly exists and that in the majority of cases it must be taken advantage of to facilitate surgical dissection of the nerve.

6. Rauber, A. A.: Nervensystem, in Rauber, A. A.: Rauber-Kopsch Lehrbuch und Atlas der Anatomie des Menschen, Ed. 11, edited by F. Kopsch, Leipzig, Georg Thieme. Callander, C. L.: Surgical Anatomy, Ed. 2, Philadelphia, W. B. Saunders Company, 1944. Strong, O. S., and Elwyn, A.: Human Neuro-Anatomy, Ed. 2, Baltimore, Williams & Wilkins Company, 1948. Clara, M.: Das Nervensystem des Menschen, Leipzig, Johann Ambrosius Barth, 1942. Polyak, S. L.: The Human Ear in Anatomical Transparencies, New York, T. H. McKenna, Inc., 1945. Grant, J. C.: An Atlas of Anatomy, by Regions, Baltimore, Williams & Wilkins Company, 1947.

We do not, however, agree as to its recognition being the rule but consider it to be only an exception.

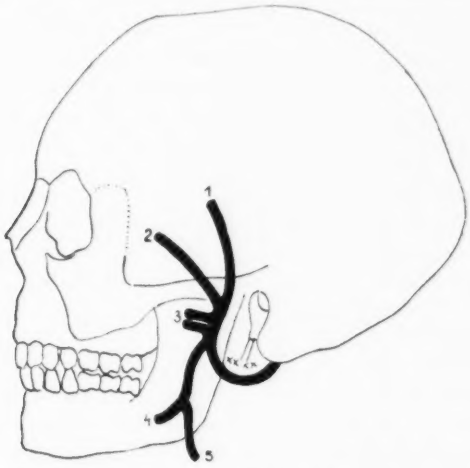
In 1946 there appeared a publication by the anatomist Hurford,[7] of King's College, London, who performed 11 dissections on cadavers and established a bilobed structure in 10 cases, with a clearly visible isthmus in 7. In all cases in which the isthmus was visible, a hard and complicated dissection was necessary. Of the 10 cases in which a bilobed structure was established, the facial nerve was placed like the meat in a sandwich in only 4 cases. In five cases one or more branches penetrated the deep lobe, and in one case several branches penetrated the superficial lobe. In 3 of the 11 cases the trunk of the facial nerve before division lay in such a manner that it penetrated one or the other lobe. In five cases the lower lobe was found to be bigger.

In Figure 1 these muscles which are important in the movements of the face from a cosmetic point of view have been rounded off. The other branches are unimportant, so that practically the surgeon need not take any notice of them when operating.

The next important question is the site where preparation of the facial nerve is to be started. It is evident that in beginning a dissection one should choose that branch which is technically favorable in thickness and constancy of position. The approach must be quick and simple and must not damage other branches or important elements. The tumor is usually just below and a little in front of the external ear and in itself is a great hindrance at the beginning of the operation. Much the same thing may be said with respect to Bailey's and Duval's methods. The upper branch of the facial nerve, which lies superficially, was selected by Sistrunk. We studied both the position and the path of the facial nerve in cadavers, and we were able to establish the following facts: The zygomatic arch is visible in most persons, and in all it is palpable, being palpable even in the thickest cheeks. Tumors are not to be found in the region of this arch, because the parotid gland almost never covers it and in the most unfavorable cases runs as far as 0.5 to 1.5 cm. below the arch and the thinned layer of the parenchyma. It is a happy circumstance that the upper branches of the temporofacial division, i. e., the temporal and especially the zygomatic branch (Fig. 1), among all the branches of the facial nerve, show the most constant position and development. These two terminal branches cross the zygomatic arch either at right angles or somewhat obliquely in the direction of the orbit. The nerves here lie directly on the periosteum. Should these two branches not be easily dissected on account of their thickness, just below the zygomatic arch and nearly parallel lies the buccal branch, which is of suitable thickness. All the other branches are inconstant in position and of varying thickness and cannot be used as a starting point for dissection. In my opinion, therefore, the zygomatic arch is the simplest and safest point of orientation, being always easy of approach and the place where dissection is always technically possible of one of the three branches, the temporal, the zygomatic, or the buccal. In no case, either in a cadaver or at operation, were we obliged to search in the parenchyma of the parotid gland.

7. Hurford, F. R.: The Surgical Anatomy of the Parotid Gland, Brit. J. Surg. **34**:186, 1946.

I have mentioned that in our method we cut the auriculotemporal nerve to sever the parasympathetic innervation, which stimulates secretion of the parotid gland. Since the experimental work of Claude Bernard, it has been known that by stimulating the chorda tympani or by cutting it, it is possible to influence the flow of secretion. This experimental fact has great significance in possible technical



1 R. temporales:	m. auricularis sup.
	m. auricularis anterior
	m. helicis minor
	m. tragicus
	m. frontalis
	m. orbicularis oculi
	m. corrugator supercilii
2 R. zygomatici:	m. orbicularis oculi
	m. zygomaticus
3 R. buccales:	m. quadratus labii sup.
	m. caninus
	m. nasalis
	m. buccinator
	m. orbicularis oris
4 R. mandibulares:	m. risorius
	m. triangularis
	m. quadratus labii inferioris
	m. mentalis
5 R. colli:	m. platysma

Fig. 1.—Topographic relation between the skull and the chief branches of the facial nerve. In the rectangle are shown the important muscles of facial expression in relation to the branches of the facial nerve.

improvements or abolition of complications in operations on the parotid gland. I have read in the literature, and have observed in our cases of post-traumatic fistulas of the parotid gland, that cutting the auriculotemporal nerve severs the secretory impulses of the gland. With enucleation or partial resection of the parotid gland there is great danger of the formation of postoperative fistulas. To

prevent these the surgeon should try to abolish secretion during the operation and, especially, during the postoperative healing process. Our chief postulate is to preserve the function of the facial nerve and to damage the salivary gland rather than the nerve.

Secretion from the parotid gland begins with dilatation of blood vessels and ceases with vasoconstriction. The nervous impulses may be not only vasomotoric but also affected through specific secretory impulses. Secretion also sets in with the contraction of the smooth muscles situated in the secretory gland, the contents of the gland being squeezed out, as Stawraky showed in 1931.[8]

The parotid gland is a purely serous gland, in contrast to the other salivary glands, which are mixed, i. e., seromucous. From the chemical standpoint the parotid gland is therefore very different from the sublingual and submaxillary glands. The vegetative innervation of the serous glands is an antagonistic one. Stimulation of the parasympathetics, i. e., the chorda tympani, results in an increase in granules in the cytoplasm and a bulging of the whole cell of the serous gland. Sympathetic stimulation, on the other hand, results in a decrease in granules and a diminution of susceptibility to stain of cell nuclei. A contrary result was obtained with the mucous glands.

Sympathetic innervation reaches the parotid gland by way of the branches of the cervical ganglia (Fig. 2). These branches, not unlike creepers, envelop the external and internal common carotid arteries and by way of nutritional arteries of the parotid gland reach the gland and perform their vegetative stimulation on the blood vessels themselves. The same process governs parasympathetic stimulation, except that the parasympathetic fibers enter the parotid gland by way of the auriculotemporal nerve. This nerve is a branch of the trigeminus, which anastomoses with the facial nerve on its trunk or its branches. Figure 3 shows the paths of parasympathetic innervation to the parotid gland. The parasympathetic fibers enter, either directly or indirectly by way of the facial nerve, the parenchyma of the parotid gland over a branch of the trigeminus; this is the auriculotemporal nerve under discussion. These terminal anastomoses take place in the region immediately before the ear.

I cannot believe that the vegetatively enervated parotid gland stops secretion permanently, an event mentioned in the literature under the heading "paralytic salivary secretion." I can well believe that secretion stops for a time sufficient for the damaged parotid gland to heal. Be that as it may, in our cases by such enervation we have obtained a clinical, operative, and curative effect.

It is known from other surgery of the vegetative nervous system that vegetative enervation is successful only if it is radical and complete. Thus it is necessary to cut all the small branches of the auriculotemporal as a secretory nerve. The presence of but a few small anastomoses renders vegetative enervation a failure.

8. Fulton, J. T.: Physiology of the Nervous System, Ed. 2, London, Oxford University Press, 1943. Tinel, J.: Le systéme nerveux végétatif, Paris, Masson & Cie, 1937. Kuntz, A.: The Autonomic Nervous System, Philadelphia, Lea & Febiger, 1947. White, J. C., and Smithwick, R. H.: The Autonomic Nervous System: Anatomy, Physiology, and Surgical Application, New York, The Macmillan Company, 1947. Pavlov, I. P.: Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex, translated and edited by G. V. Anrep, New York, Oxford University Press, 1927. Gantt, W. H.: Recent Work of Pavlov and His Pupils: Conditioned Reflexes; Sympathetic Nervous System (Orbeli); Epilepsy and Cerebrospinal Fluid (Speransky), Arch. Neurol. & Psychiat. **17**:514, 1927.

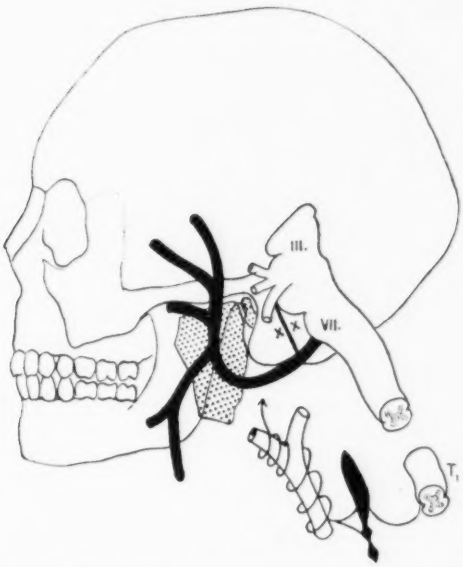


Fig. 2.—The skull, with the facial nerve, parotid gland, carotid bifurcation, brain stem, medulla oblongata, the thoracic section of the spinal cord, and the sympathetic and parasympathetic nervous system; *x.x* indicates the auriculotemporal nerve.

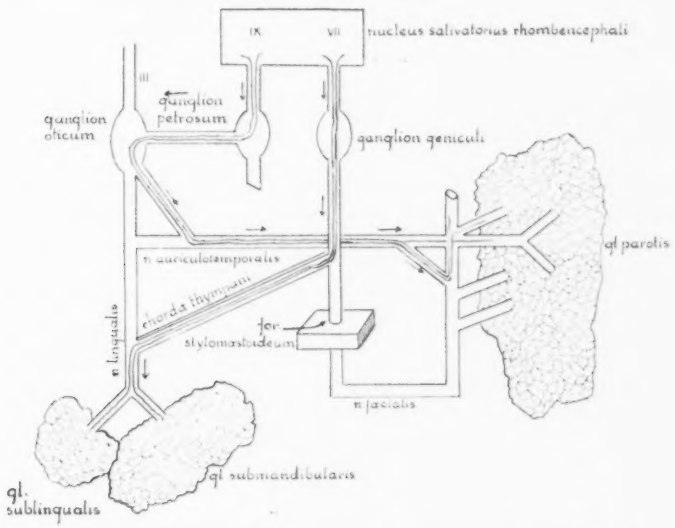


Fig. 3.—Graphic presentation of the parasympathetic innervation of the salivary glands. The parasympathetic secretory fibers use three nerves—the trigeminal, glossopharyngeal, and facial—to reach their destinations, the salivary glands, from a common center, the nucleus salivatorius rhombencephali. The parotid gland receives fibers via the glossopharyngeal nerve, the otic ganglion, the petrosal ganglion, the trigeminal nerve, and, finally, the auriculotemporal nerve. The submaxillary gland receives fibers via the facial nerve, the geniculate ganglion, the chorda tympani, and the trigeminal nerve.

The auriculotemporal nerve appears in the region of the parotid gland behind the neck of the articulation process of the mandible. In our dissections we could see three kinds of terminal branches (Fig. 4). One kind entered the trunk of the facial nerve and took this nerve as a path to the parotid gland. In our opinion one ought to call this branch or branches the anastomotic part of the auriculotemporal nerve. In our operative material as well as in our studies on cadavers, we saw this anastomosis with the facial nerve as one, two, or three branches or as a subtle plexus. Such anastomoses often appear at the beginning of the temporofacial division of the facial nerve close behind the division and more rarely at the trunk itself. The second type of branch shows no anastomosis with the facial nerve but sinks independently into the parenchyma of the parotid gland. It is the

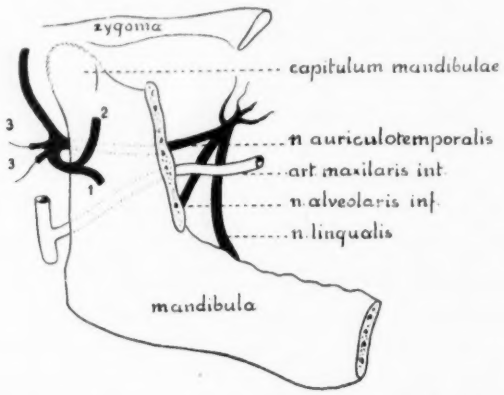


Fig. 4.—The lower jaw, zygomatic arch, and cartilaginous ear funnel, with the auriculotemporal nerve. This nerve comes from the depth between the joint of the lower jaw and the ear funnel. The text describes three kinds of terminal branches of the auriculotemporal nerve.

most capricious type in number and thickness. The third kind of small branch is in reality the prolongation of the auriculotemporal nerve toward the planum temporale or the external ear, which it innervates. The auriculotemporal nerve is constant in its basic position, being a branch of the trigeminus. As Figure 4 shows, it leaves the trigeminus and enters the space close behind the joint of the mandible, formed on one side by the ligamentum sphenomandibulare or the mandible. It is a mixed nerve, i. e., a nerve of sensation with parasympathetic fibers to stimulate salivation. When operating, we perform enervation simply by cutting all the nerves which approach the parotid gland or the facial nerve from the space formed, on one side, by the edge of the mandible and the neck of the articulation process of the mandible and, on the other, by the cartilaginous funnel of the external auditory channel.

Taking into consideration the literature as well as our experiences and findings, I should like to summarize the aspects of surgical anatomy that I think are important:

1. The bilobed structure of the parotid gland is evident in the majority of cases and plays a certain role in dissection of the facial nerve and its branches.

The relation of the trunk of the facial nerve to the interlobal plane and, especially, the relation of the branches of the facial nerve to the plane in most cases are not such as to favor an operative technique founded on the interlobal position of the trunk and its branches. In spite of the bilobed structure and interlobal position of the facial nerve, dissection of this nerve from the parotid gland remains hard work which requires a safer starting point than removal of the upper lobe.

2. The zygomatic arch, which is not covered by the parenchyma of the parotid gland, constitutes the easiest-found point of orientation for dissection of the constantly placed branches of the facial nerve, which, owing to their thickness, warrant their localization and subsequent dissection in a central direction.

3. The vegetative secretory enervation of the parotid gland is performed by cutting all the terminal branches of the auriculotemporal nerve. Practically, it is necessary for the surgeon to divide these terminal branches into (1) those which are in direct anastomosis with the facial nerve, (2) those which enter the parotid gland directly, without anastomosis with the facial nerve, and (3) those which are spread as nerves of sensation either upon the planum temporale or upon the region of the auditory channel. Cutting these branches does not injure the patient either cosmetically or as to his general condition.

OPERATIVE PROCEDURE FOR EXTIRPATION OF PAROTID GLAND TUMORS: PARTIAL RESECTION AND TOTAL ABLATION OF THE GLAND

The operative procedure presented was used in the case of tumors that had been diagnosed from the clinical picture and puncture biopsy as benign and without signs of infiltrating growths. We do not think it advisable to use this method for malignant tumors infiltrating into the nerve and surrounding tissues. For such tumors the old surgical rule is valid: "Resect the tumor beyond its actual boundary irrespective of cosmetic or functional consequences and extirpate the regional lymph nodes." For malignant tumors in the parotid region the great majority of surgeons agree also upon conservative radiological treatment. We must admit that, in spite of the most conscientious clinical examinations and biopsies, mistakes are liable to occur; for example, when in another part of the tumor a malignant alteration has taken place. In such cases our procedure would still be indicated, but operation should be followed by x-ray therapy.

In our procedure the patient lies on his back with his head turned in the direction opposite to the tumor. It is best to fix his head by means of an adhesive tape fastened around the headrest. Our first patients were operated upon under local infiltrative anesthesia involving the whole preauricular region as well as the retroauricular space. The infiltration spreads toward the neck and mandible. We do not consider it necessary to add epinephrine to the usual 0.5% solution of procaine hydrochloride to diminish the parenchymatous hemorrhage. Procaine hydrochloride is sufficient for a clear and easy preparation of the small branches. In the last third of our cases intratracheal oxygen-thiopental-ether anesthesia was used. This kind of anesthesia also proved the best in other cosmetic and prolonged operations. The exact and painstaking work of preparation requires a good nervous condition on the part of the surgeon, who must not be disturbed by restlessness of the patient.

The operative procedure is divided into six stages.

1. *Cosmetic Skin Incision* (Fig. 5*A*).—The incision is given the shape of an inverted "W" situated in the region immediately before and below the ear lobe. One of its terminal wings is placed along the zygoma and the other along the anterior edge of the cranial third of the sternocleidomastoid muscle. The zygomatic wing of the incision should be made only as deep as the subcutis, while the middle of the cut, which goes around the lower part of the ear lobe, should reach the perichondrium of the ear funnel. The lower wing of the cut frees the anterior edge of the sternocleidomastoid muscle as well as the upper surface of the mastoid process. During this part of the procedure the surgeon cannot hit the facial nerve, and yet he creates an entrance into the operative field.

2. *Exposing the Cartilage Funnel* (Fig. 5*B*).—With a finely constructed periosteum elevator it is easy to dissect the whole of the cartilage funnel as far down as the temporal bone. This act frees the tumor and the parotid gland from the ear and creates an exceptionally wide approach to the operative field. It offers no possibility of collision with the trunk or branches of the facial nerve whether these are normally placed or pathologically displaced. The wings of an écarteur placed between the cartilage funnel and the edge of the wound make the latter gape to display the cartilage funnel in its entire depth.

3. *Preparation of the Orbicular Branch of the Facial Nerve* (Fig. 6*A*).—This stage of the operation should be performed with special care and patience and without haste. Preparatory work should be done with a fine dissector and scissors. It is always possible eventually to find the fine orbicular branch, which, being closely adherent to the periosteum of the zygoma, crosses the zygomatic arch obliquely (Fig. 6*A*). Should the surgeon experience difficulty in finding this branch, he should locate the middle branch, which is the strongest one immediately below the zygomatic arch and closely parallel to it. Its course is parallel not only to the zygomatic arch but also to the transverse facial artery. On the zygomatic arch the surgeon must find the orbicular branch. The arch is covered only by skin and fatty subcutis. The orbicular branch is situated immediately above the periosteum of the arch. It is true that here one can find small arterial and venous branches running toward the planum temporale, but they are easily differentiated from the nerves. The nerve is dissected and prepared by dividing the tissue with scissors, but only along the nerve—a procedure preventing the nerve from being injured. The parenchyma of the parotid gland is never found above the arch. Once the orbicular branch has been located, the surgeon must proceed deeper, only to find that it disappears in the tissue of the parotid gland. The branches of the preauricular nerve, on the other hand, disappear in the subcutis.

The branch found, the whole procedure that follows is but a matter of patience. The surgeon need not fumble in the dark, as everything that follows is well planned and has its well-founded orientation.

4. *Preparation of the Facial Nerve Through the Parotid Parenchyma or Tumor; Removal of the Tumor and Parotid Gland* (Figs. 6*B* and 7*A*).—Having found the aforementioned elements, the surgeon starts preparation of the orbicular branch in a central direction without taking notice of possible damage to the gland or tumor. Primarily his attention is directed to the nerve; that is, he performs dissection,

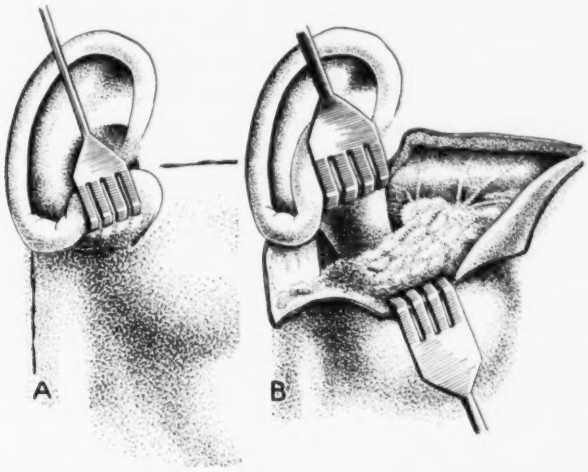


Fig. 5.—*A*, first stage of the operative procedure: cosmetic skin incision in the shape of an inverted "W" before and below the ear lobe. *B*, second stage of the operative procedure: exposure of the cartilage funnel as far down as the temporal bone, creating an exceptionally wide approach to the operative field, tumor, and parotid gland. The upper terminal wing of the incision placed along the zygoma creates an approach to the orbicular branch of facial nerve, which can without exception be found closely adherent to the periosteum of the zygoma.

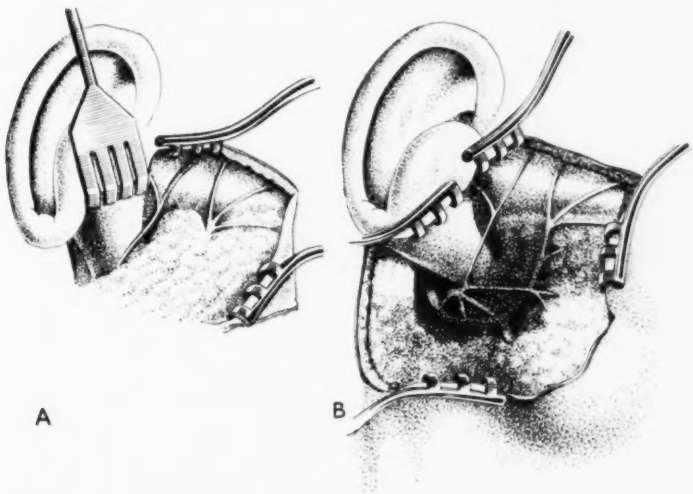


Fig. 6.—*A*, third stage of the operative procedure: preparation of the orbicular branch of the facial nerve, which is above the zygomatic arch. In the upper incision is seen the terminal ramification of the orbicular branch of the facial nerve (temporal, zygomatic, and buccal branches). This ramification is always closely adherent to the periosteum of the zygomatic arch. The temporal branch lies at an oblique angle, the zygomatic at a right angle, and the buccal parallel below the zygomatic arch. Parotid parenchyma always stops like a flat cake on the lower edge of the zygomatic arch. This fact insures a safe starting point for preparation. *B*, fourth stage of the operative procedure: preparation of the facial nerve through the parotid parenchyma or tumor. The text describes this stage in detail. Cutting the anastomoses between the facial and trigeminal nerves and the auriculotemporal nerve concludes the fifth stage of the procedure.

cutting, or morcellement of the surrounding tissue toward the pes anserinus of the facial nerve. The nerve should never be touched by the forceps; it should be removed with a blunt dissector while it is supported by blunt hooks. In following the dissection mentioned the surgeon will never have to complain of the slightest or most transient paresis. The nerve is vulnerable to squeezing, cutting, or distending but not to displacing. The success of the operation depends upon this handling. Even the best-prepared nerve is liable to palsy if handled roughly.

Having reached the pes anserinus by dissection, the surgeon will locate the other branches of the facial nerve more easily because of their thickness. Everything that lies between the pes anserinus and the cartilage funnel may be cut without damage to any structure anatomically important. Such a cut in the direction of the car-

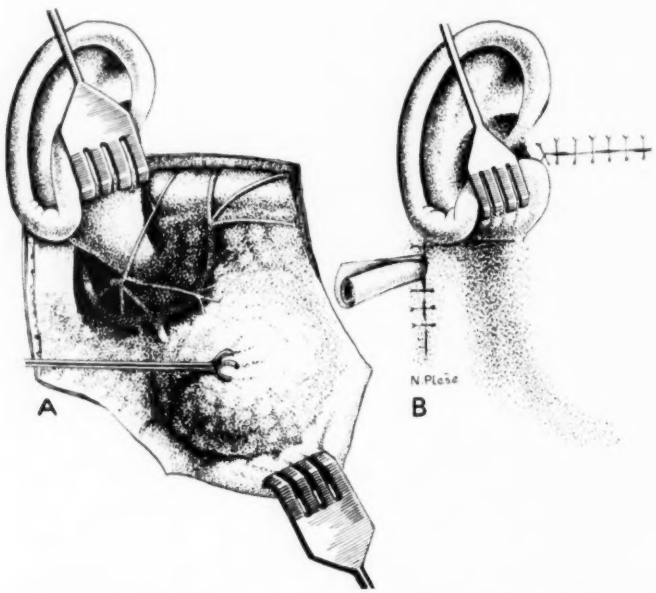


Fig. 7.—*A*, removal of the tumor and parotid gland. The tumor is removed by (1) simple enucleation, (2) partial resection, or (3) morcellement. *B*, closure of the wound with the drain in place.

tilage funnel gives a full view over the operative area and, moreover, mobilizes the tumor, which now remains with the parotid gland lying upon the skin and fascia. On entering the area of the tumor, the surgeon does not take any notice either of the tumor or of the parotid gland except to isolate the nerve from this mass. It may be that the pes anserinus is not fully developed anatomically, in which case one should follow the branches to reach the usual peripheral branching of the facial nerve. The trunk of the facial nerve need not lie between the two lobes of the parotid gland. Usually the division is invisible. Often the branches of the facial nerve pierce the mass of the tumor, so that it is necessary to cut the tumor in order to isolate the nerve. This is an especially frequent occurrence in the lower branch of the facial nerve.

The procedure described leads to a phase of the operation in which the nerve is freed from both the parotid gland and its tumor, and the whole procedure, open to view, is simple and self-evident. In case the tumor is well bordered the surgeon restricts himself to the least possible damaging of the parotid gland. However, in case of the slightest uncertainty as to its borders, he removes all of the gland that is visible. If the tumor is adherent to the sternocleidomastoid muscle he has to sacrifice the muscle to the extent necessary for a complete removal of the tumor. Damage to the blood vessels or nerves in this operative area will have no ill effects; preservation of the facial nerve is all-important.

5. *Cutting the Auriculotemporal Nerve* (Figs. 6*B* and 7*A*).—After the procedure described the facial nerve will lie exposed in the operative field like an anatomical preparation, so that it can be followed with ease as far as its entry into the stylomastoid foramen. In the direction of the cartilage funnel will be seen either a series of nerve anastomoses or one thick anastomotic branch. These belong to the system of anastomoses between the facial and trigeminal nerves, i. e., to the auriculotemporal nerve, which is responsible for the secretory innervation of the parotid gland. By cutting this system one severs this innervation and prevents—especially with incomplete ablation of the gland—unpleasant salivary fistulas. Practically all the branches of the facial nerve going toward the ear lobe must be cut to insure elimination of the anastomoses between the trigeminal and auriculotemporal nerves to eliminate parasympathetic secretory impulses.

As has been stated, not all the terminal branches of the auriculotemporal nerve anastomose with the facial nerve; some run directly into the parotid gland. These branches are cut by the aforementioned technique (Fig. 7*A*). The tumor is removed by (1) simple enucleation, (2) partial resection, or (3) morcellement. Enucleation is performed in cases of well-bordered tumors, the preparation of the nerve being a safeguard against lesions and fistulas. Partial resection is indicated when the borders of the tumor are not pronounced. In such cases the tumor and parotid parenchyma after the preparation of the facial nerve are not infrequently situated above the nerve and its branches. Partial resection of both gland and tumor is then required. The technique described constitutes a safety measure against the occurrence of salivary fistulas. Where the prepared nerve is embedded in the tumor itself or lies inside or beneath the tumor or the parotid gland, morcellement of the tumor and gland is indicated.

6. *Closing and Draining of the Wound.*—Closing the wound is an easy procedure. The edges are subcutaneously sutured with catgut along their entire length. Then cutaneous suturing is done with finest silk. The tension of the sutures should not be greater than to secure a mere adhering of the skin edges. The retroauricular part of the cut is left open 1 cm. A rubber cigarette roll made of worn-out rubber glove is then inserted into the wound (Fig. 7*B*). This roll insures good drainage, while its softness prevents decubitus of the tissue and the laboriously prepared nerve.

Forty-eight hours after the operation the drain is removed; the wound heals within another three to four days. The sutures are removed the third day after operation. Lack of sensation in the ear lobe lasts about two months. Paresis (but never palsy) is present only in the upper branch of the facial nerve for the first few weeks after surgery.

OPERATIVE MATERIAL

We performed this operation in 43 cases. Figure 8 shows the sex and age of the patients. The pathological-histological diagnosis was given us by the Pathological Institute of the Medical Faculty of Zagreb (Prof. Dr. Saltikov).

The aim of this paper is not to deal with a complicated, noncrystallized, and even problematical nomenclature and classification of tumors of the parotid gland. Classification does not influence our decision or our method. In addition to the cases summarized in Figure 9, we observed other cases in which we did not perform the operation or in which the growth was a large malignant tumor extending into the surrounding tissue or a "tumor" clinically diagnosed as tuberculosis or actinomycosis. We performed preoperative diagnostic puncture biopsies and examined the tumors histologically. We did not get the impression that these procedures provided a more accurate differential diagnosis between what was benign and what was malignant. In some cases puncture biopsy revealed a benign tumor and subsequent examination of the extirpated tumor disclosed a malignant alteration in several parts. On the other hand, a diagnosis of malignancy was established in cases in which there was little or no clinical evidence of malignancy because the tumor was movable and clearly defined. In spite of the diagnosis of malignancy we did not alter our determination to operate by my method. We firmly maintain, however, that obviously malignant tumors infiltrating the surrounding tissue as well as the facial nerve cannot be the subject of a method the aim of which is to preserve the nerve. We have no intention of leaving the nerve in a state of infiltration by malignant cells. In such cases we consider my method, but only combined with x-ray therapy.

We do not let results of biopsy determine our decision in favor of or against operation. Our material shows that the branches as well as the stem of the facial nerve run through the tumor or that the branches are displaced by distention in such a manner that we are positively against any attempt at a probatory excision. Figure 9 illustrates a case in which the trunk of the nerve was cut out of the tumor mass and the groove of the prepared nerve was visible. Even puncture biopsy with a thick needle seems to us to be hazardous. We do not like to perform a puncture biopsy when tuberculosis, actinomycosis, or a retention cyst due to sialolithiasis is suspected. We do not consider sialography a satisfactory diagnostic method in cases of parotid tumor.

In all our cases presented in Figure 8 the tumor was situated so as not to cover the zygomatic arch. This circumstance made the search for the upper branch of the facial nerve easy, and this branch constituted a safe guide at preparation. As already stated, all the tumors in our cases were below and a little behind the ear lobe, or below and a little before it, but always so placed that they did not cover the zygoma. This is important, because we decided on principle to prepare the facial nerve in the region of the upper, easily approachable branch instead of the trunk or lower branch, which, in our material, was always covered either by the gland or by the tumor. It will not be superfluous to mention again our experience showing that the nerve or its branches ran through the tumor and lobes of the bilobed parotid gland. Likewise, there existed not infrequently marked dystopies of the branches brought about by the growth of the tumor, so that any other approach to preparation of the nerve might have been hazardous.

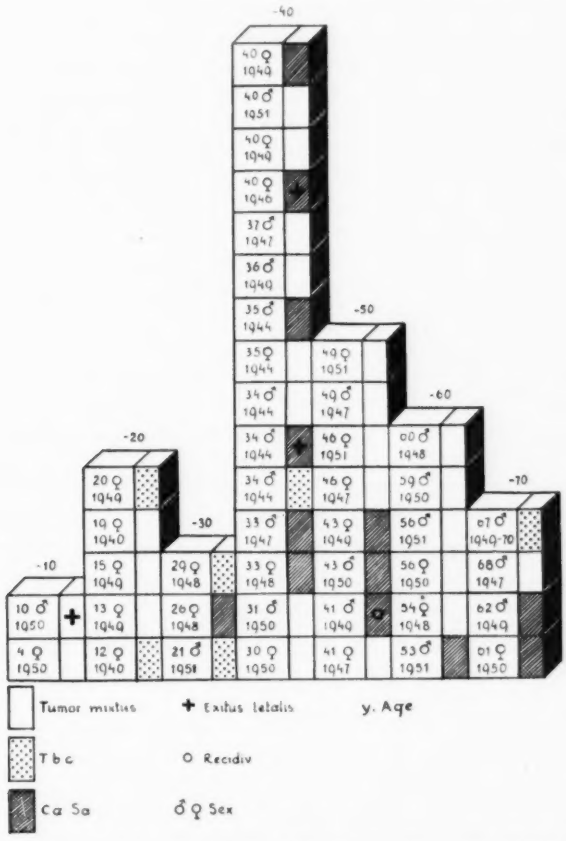


Fig. 8.—A chart summarizing 43 cases in which the operation was performed between 1940 and 1951.

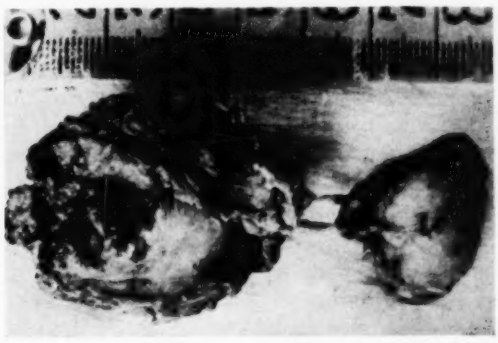


Fig 9.—Enucleated mixed tumor of the parotid gland. This tumor shows two peculiarities: It is a double tumor connected by fibrous adhesions, and the larger part was pierced by the trunk of facial nerve. The groove from which the trunk of the facial nerve was cut out can be seen.

During our preparation of the nerve, parotid gland, and tumor, we discovered only in two cases a pronounced bilobed structure of the gland in Bailey's sense. Despite lack of experience we do not doubt that it would be easier in many instances to discover the bilobed structure by preparing the nerve from below upward instead of from above, but we do not believe that the tumor growth would leave this structure visible. It is possible that the bilobed structure is more easily identified in normal parotid glands than in glands containing tumors, where the parenchyma is changed by force of an expansive or infiltrative growth of the tumor (note the experience of Hurford [7]).

Only in 2 of our 43 cases could we blame our procedure for the occurrence of palsy. One patient showed evidence of paresis before the operation, though in a lesser degree than after the operation. The tumor was suspected of malignancy and was later identified as a sarcoma. The facial nerve recovered function half a year later, which means that it was damaged by difficult preparation and not by the supposed infiltrative growth of the tumor. The other patient showed a transitory paresis of the whole facial nerve, but at the first checkup a month after operation the palsy was no longer present. The tumor in this case was histologically a "mixed tumor of the parotid" the size of a fist. The branches of the facial nerve lay in front and behind it, making preparation very difficult, and we believe that the transitory palsy was brought about by distention.

In all our cases we could observe in the first days after the operation some weakness in moving the brows and forehead as well as a certain difficulty in closing the eyelids on the side of operation. All patients, however, assumed quite a normal facial expression afterward. Such accidents are the sequelae of unavoidable maltreatment during the preparation of the thin orbital branch.

Concerning the salivary secretion of the remaining parts of the parotid gland as well as that of the fistulas, the following statements are relevant. Total ablation was carried out only in one fourth of our cases. In the remaining cases we performed either resection of the parotid or enucleation of the tumor. It is evident that by our procedure the parenchyma of the parotid gland is always damaged, an accident hardly to be avoided. In spite of cutting of the auriculotemporal nerve, one patient with incomplete ablation of the gland had a 20-day postoperative salivary secretion; the presence of diastatic ferment in the secretion was biochemically established. All the other patients showed a normal wound secretion, the wound healing in a few days. The occurrence of a 20-day secretion is not, in our opinion, likely to happen again. We were misled by incomplete preparation of the trunk of the facial nerve so that no radical cutting of the anastomoses between the facial and auriculotemporal nerves was performed (as mentioned in the first part of my paper). The incident happened because the well-bordered tumor had sprung out and we thought it possible to shorten the time of operation. In short, we did not cut all the secretory nerves. The salivary secretion always started at meals and flowed out through the drain behind the ear lobe.

I used the same surgical procedure in four cases of post-traumatic salivary fistulas, in three of which it was successful. The failure was due to a wide, hard scar which prevented radical cutting of the anastomoses between the facial and auriculotemporal nerves.

The subject of surgical treatment of post-traumatic parotid fistulas will be dealt with elsewhere. I have mentioned these cases only to verify the possibility of influencing salivation by my method, which can be carried out most safely by finding the upper branch of the facial nerve.

The same method of preparation and the same surgical procedure were applied in one case in which neurolysis of the facial nerve was performed in order to eliminate palsy due to strangulations. (The same patient was operated upon for a tumor at another clinic.) The palsy disappeared in three weeks.

Our cases are grouped according to age decades in Figure 8, with sex, age, year of operation, kind of tumor, sign of recurrence, or death indicated. Thus the chart shows the relation between permanence of the operative results and these factors.

Characteristic was the incidence of malignant alteration occurring from the second decade onward. The incidence of tuberculomas was relatively high. These tumors did not show macroscopically any symptoms suggesting tuberculosis before, during, or after operation. A rather high frequency of parotid tuberculomas might be attributed to the high incidence of tuberculosis in our country. Of the 43 patients, 2 died two years after operation and 1 the day after operation, while all the others except one—who showed marked symptoms of a recurrence—are alive and well. One of the patients who died two years after operation had a carcinoma and the other a sarcoma. The former died of cerebral metastases and the latter had packet-like metastases on the neck before death, which occurred outside the hospital. The patient showing a recurrence was later operated upon for carcinoma; an ulcerative infiltration the size of his palm developed afterward behind and below the ear lobe. He was being given x-ray treatments at the time of this writing. All the patients whose tumors showed histological evidence of malignant alteration were subsequently treated by x-rays.

The third death was that of a 10-year-old boy with a big tumor with an indefinite border. He suffered profuse hemorrhage during operation, and it was necessary to give him a blood transfusion. He recovered from the shock, but the next day he suddenly died. Postmortem examination provided no explanation as to the cause of death. Histologically, the fibrous tissues of the tumor contained cartilaginous islands with hemangiomatous tissue and nervous elements. No evidence of malignancy was present.

Thus, among our 43 cases there were only two transient palsies, three deaths, and one local recurrence. No recurrence of the benign tumors could be observed.

The material described, which was selected for my method of operation on the assumption that all the tumors were benign, shows that such tumors are indeed on the borderline between benignancy and malignancy. For this reason I maintain that operation should be performed as soon as the tumor appears. My successful procedure will not prevent me in the future from making a quick decision in favor of a radical operation from fear that I may either damage the facial nerve or produce unpleasant fistulas and thus disfigure the patient.

SUMMARY

I have presented the experience of myself and my associates with radical operation for tumor of the parotid gland and have proposed a method of my own.

The surgeon's dilemma consists in the fact that he is at a loss as to a decision regarding so-called "mixed tumors of the parotid," which are on the borderline between benignancy and malignancy and for which operation represents a possibility of damage to the facial nerve and production of unpleasant fistulas. The search for a system of operation in the field of the parotid gland is described. The aim of such a system is to provide the surgeon with a means of insuring the usual surgical safety.

What is fundamentally new in my method is as follows: 1. The special circumauricular skin incision is cosmetically excellent owing to its liberating the cartilage ear funnel from the parotid gland and tumor. 2. A certain and safe discovery of the upper branch of the facial nerve is insured, this branch being my guide in the dissection of the nerve from the parotid gland and tumor. 3. The cutting of the anastomoses between the facial and auriculotemporal nerves, the latter being a salivary secretory parasympathetic nerve, renders formation of postoperative fistulas impossible; this addition to the operation is performed routinely; it makes possible preparation of the facial nerve without regard to possible damage to the parotid gland.

In connection with presentation of my method, an extensive description is given of the anatomy and physiology of the facial and auriculotemporal nerves and of the parotid gland. My method is recommended on the basis of an experience including 43 cases of total ablation, partial resection, or enucleation of the parotid gland or tumor. In only two cases was there a transient palsy. Our material was selected for this operation on the assumption that in all cases the tumor was benign. Figure 8 indicates that parotid tumors are on the borderline between benignancy and malignancy. For this reason I maintain that they should be operated upon as soon as they appear.

BENIGN PAPILLOMA OF THE PERITONEUM

Report of a Case (Incident to Surgery for Removal of Adenomatous Polyp of the Sigmoid Colon) and Review of the Literature

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BENIGN papilloma of the peritoneum is a rare lesion, seldom noted except incidental to laparotomy or to postmortem examination. In fact, it is improbable that a diagnosis of this lesion can be made except by direct visualization of the peritoneum.

A review of the literature reveals confusion with regard to this tumor and the more commonly reported mesothelioma of the peritoneum to which it bears a poorly understood relationship. So far as can be reasonably determined, this lesion was first recognized by Wells,[1] in 1935, who described the tumor as a benign, branching villous papilloma. The lesion was noted as an incidental finding at autopsy of a 79-year-old man whose death was secondary to suprapubic prostatectomy. Wells found this tumor to consist of numerous villous processes, covered by cuboidal cells continuous with the mesothelium of the peritoneum; the stoma was coarse, composed of connective tissue, and multiple lesions were noted on the inferior portion of the diaphragm, the omentum, and the mesentery. The minute villous processes contained no evidence of mitoses or other malignant characteristics, as illustrated in the photomicrographs.

In contrast with this rare peritoneal papilloma, which apparently has been reported only once previously in the literature, one finds isolated reports of mesothelioma or "endothelioma," which on close scrutiny possess both the clinical and the histologic attributes of malignancy.

The subject of mesothelioma was reviewed by Ramsey and Chomet [2] in 1943. These authors, in reporting their own experience and in reviewing the few cases reported prior to that time, stressed the uncertain classification and meager understanding of these lesions. They pointed out that mesotheliomas have mitoses, may produce mucin, and frequently possess reticulum-positive stroma. Clinically the

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1. Wells, A. H.: Papillomatosis Peritonei, Am. J. Path. **11**:1011-1015 (Dec.) 1935.

2. Ramsey, T. L., and Chomet, B.: Mesothelioma of Peritoneum, Arch. Path. **35**:292-298 (Feb.) 1943.

behavior in this series of cases was progressive and fatal. Rhind and Wright,[3] in 1949, described a case of mesothelioma with clinically and histologically malignant characteristics occurring in a 20-year-old man. A similar case was reported by Mathews [4] in 1950. From a survey of the half-dozen cases in the literature, the male sex appeared to predominate and the age group to cover a wide span, from the second to the sixth decade, although with respect to these factors no significant conclusion is possible.

The mesothelioma appears to be a malignant tumor, having pericardial and pleural counterparts, and constitutes a primary cause of death, as distinct from the benign papilloma. Thus it would appear possible to attempt classification of those tumors derived from the serosa of the peritoneum. Provided the previously mentioned criteria are present, it is possible to separate benign papilloma from malignant mesothelioma. In addition, since the peritoneum is mesodermal in origin rather than endothelial, the term mesothelioma appears more descriptive than does the term endothelioma.

With regard to the origin of benign peritoneal papilloma, it can be theorized that the papilloma represents (1) implants from a ruptured mesothelial cyst, (2) proliferative papillomatosis of connective tissue origin, not necessarily constituting an endothelial or mesothelial neoplasm, or (3) embryonic derivatives of peritoneum arising from ectopic displacement of cells from the genital ridge in the earliest weeks of fetal development.[5]

To the surgeon or pathologist other peritoneal tumors or tumor-simulating conditions may grossly resemble the benign papilloma. In the benign group are angioma, lymphangioma, lipoma, leiomyoma, corpus liberum, pneumatosis cystoides, endometriosis and other chronic inflammatory lesions. In the malignant group are myxoma peritonei, metastatic and secondary carcinoma, sarcoma, lymphoma, and mesothelioma.[6]

Treatment of benign papilloma of the peritoneum, when localized, as in the case presented, should consist of surgical excision whenever practical, because the diagnosis is made in retrospect by pathologic examination and because of the possibility of sarcomatous degeneration in benign tumors of mesenchymal origin.

REPORT OF A CASE

E. C., a 39-year-old male physician, was admitted to the proctologic service of Temple University Hospital on Sept. 1, 1949, with the complaint of having noted small amounts of bright-red blood accompanying otherwise normal bowel movements, occurring intermittently for a six-month period. Prior to this he had been in excellent health. System review, past history, and family history were noncontributory. Physical examination, including proctosigmoidoscopy, revealed no abnormal findings. However, special roentgen studies of the lower bowel, using an opaque medium, demonstrated a polyp-like defect of the sigmoid colon. All laboratory findings, including prothrombin time, chest x-ray study, fasting blood sugar, and

3. Rhind, J. A., and Wright, C. J. E.: Mesothelioma of Peritoneum: Report of Case and Review of Literature, Brit. J. Surg. **36**:359-363 (April) 1949.

4. Mathews, W. H.: Case of Peritoneal Endothelioma, Canad. M. A. J. **62**:376-379 (April) 1950.

5. Arey, L. B.: Developmental Anatomy: A Textbook and Laboratory Manual of Embryology, Ed. 4, Philadelphia, W. B. Saunders Company, 1941, pp. 265, and 281.

6. Karsner, H. T.: Human Pathology, Ed. 7, Philadelphia, J. B. Lippincott Company, 1949, p. 541. Moore, R. A.: Textbook of Pathology, Ed. 2, Philadelphia, W. B. Saunders Company, 1951, p. 852.

electrocardiogram, were within normal limits. After preparation for intestinal surgery, laparotomy was performed on Sept. 8, 1949, the abdomen being entered through a left paramedian muscle-retracting incision. A movable polypoid mass approximately 1 cm. in size was palpated within the lumen of the sigmoid colon, having a limited range of excursion. A solitary plaque 0.5 cm. in diameter having the characteristics of a cyst was noted on the anterior surface of the left lobe of the liver. On initial exploration of the abdomen there were no other abnormalities immediately apparent, with the exception of the two above-mentioned findings. The sigmoid colon was isolated by moistened laparotomy sponges, and through a longitudinal incision into the sigmoid, the polypoid mass was delivered, its pedicle clamped, divided, and sutured with 0 chromic absorbable surgical (gut) sutures. No other intraluminal lesions of the contiguous bowel were present, and the sigmoid incision was repaired by continuous 0 chromic gut sutures, with reinforcing 000 nonabsorable surgical (silk) interrupted sutures in the serosa. After this, a soft irregular polypoid mass was noted near the sacral promontory on the right side of the sigmoid mesentery, contiguous with the peritoneum and approximately 1 by 2 cm. in size. It was dissected and excised and the peritoneal defect repaired with 0 chromic gut sutures. A sump drain was placed on the pelvic floor and the abdomen closed, using interrupted figure-of-eight No. 30 steel-wire sutures for peritoneum and fascia; a continuous No. 35 steel-wire suture was used for skin closure. Streptomycin (1 gm.) and penicillin (500,000 units) were instilled through the sump drain. The patient was returned to his room in good condition. The sump drain was removed the following day and, except for a transient episode of atelectasis, uneventful convalescence ensued, the patient being discharged from the hospital on the 10th postoperative day.

The patient has remained well and free of any recurrence almost three years following excision of peritoneal papilloma incidental to transsigmoidal polypectomy.

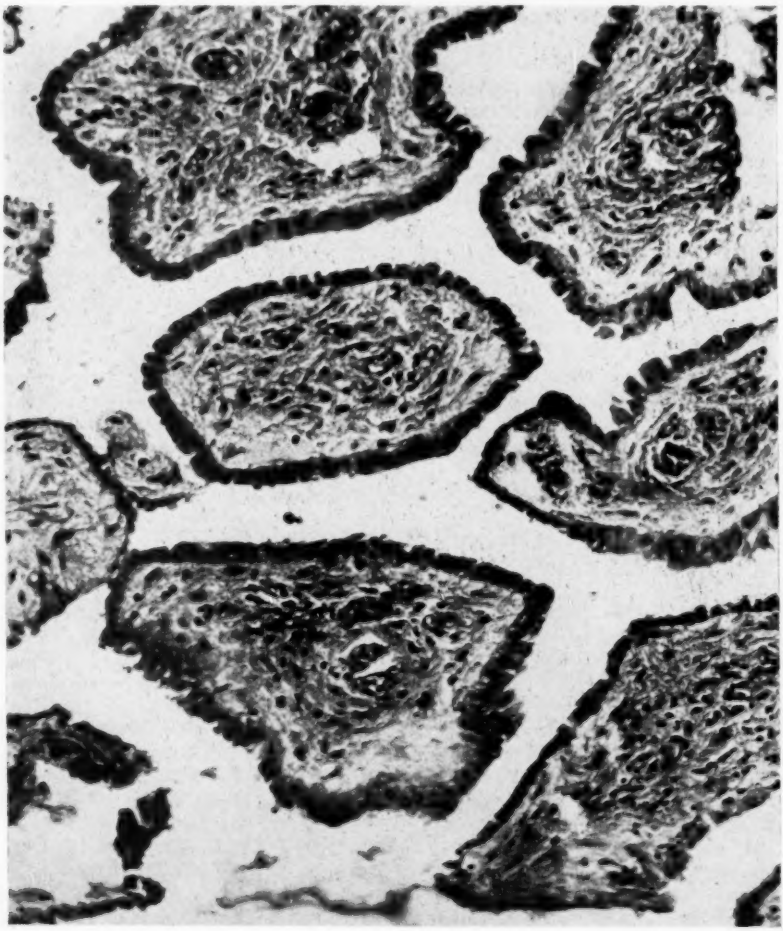
Pathologic Study.—The following pathologic report was made concerning the specimens removed. The sigmoid polyp consisted of a benign adenoma with pedicle. The peritoneal lesion, when sectioned, was found to consist of numerous villous stalks of a loose aureolar tissue covered by a single layer of cuboidal cells (Figure). The latter had the same appearance as those found in chronic though mild inflammatory states of the peritoneum. Their arrangement on the surface appeared to be quite orderly, although there was little evidence of a basement membrane. The nuclei were rather large and spheroid with considerable rather loose chromatin. None of these cells had the goblet shape and the "dropping-off" appearance that characterizes many of the malignant mesotheliomas. There was no cytoplasmic vacuolization suggesting phagocytosis or mucin secretion.

The supportive core consisted of a very loose arrangement of polyhedral spindles or stellate cells in a myxomatous matrix. The nuclei of many of these cells had the morphologic characteristics of those in the covering layer and one obtained the impression that the two types of cells were transitional forms of each other. The stroma was rather vascular. Some of the vessels were small with well-formed walls; others were much larger with walls of greatly extended endothelial cells. There appeared to be some irregular lymphatic spaces. There were a few small foci of lymphocytes and an occasional neutrophile suggesting a mild inflammatory reaction. All the cells of this structure had a morphology consistent with a mesothelial origin. Scrutiny of the photomicrographs illustrated in Wells's case reveals the lesions of the two cases to be almost cytologically identical. One of us (E. A.) has seen the sections from two other rather similar cases. Both the latter lesions were found in children incident to herniorrhaphy. Both had certain features which set them apart from the lesion described above. The covering cells in one were more frequently cylindrical than cuboidal; in many areas of both the covering was multilayered and in the stromal cores there were spheroid laminated bodies resembling the psammoma bodies of meningioma.

COMMENT

To date, none of the lesions with which we are familiar have demonstrated the progressive growth which is essential to the character of a true neoplasm. One should be cautious about considering these as such since we are fairly familiar now with several similar papillary lesions of the skin and mucous membranes which are manifestations of virus infection. Until more is known of the ultimate behavior of

these rare and curious lesions, it is probable that any positive statement made concerning them should be made with reservation. Their relationship to malignant mesothelioma is poorly understood. At present it can be concluded that the origin of peritoneal papilloma is from mesothelium and that the lesion appears to run an innocuous course.



Photomicrograph showing cross sections of numerous villous processes, each with a fibrous stromal core bearing the blood supply to the process. Each is covered by a single layer of cuboidal mesothelial cells. Hematoxylin and eosin stain; × 200.

SUMMARY

Benign papilloma of the peritoneum is a rare lesion, believed to have been reported only once previously and first recognized as such by Wells in 1935.

Features are presented upon which, if present, a differentiation can be made between benign papilloma and malignant mesothelioma, which is more commonly reported and to which papilloma may bear a poorly understood relationship.

The origin, diagnosis, treatment, and prognosis of benign peritoneal papilloma are discussed.

The successful removal of a benign peritoneal papilloma from a 39-year-old man, a physician, is reported, incidental to surgery for extirpation of a benign adenomatous polyp of the sigmoid colon. The patient is alive and without symptoms or recurrence three years following surgery.

The histopathology of this lesion is described, illustrated by a photomicrograph, and a review of the pertinent literature is presented.

It is hoped that this report will aid in the further understanding of the lesion, the incidence of which is believed commoner than indicated by the small number of cases reported in the literature.

POLYETHYLENE ABDOMINAL PACK

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INTRAPERITONEAL adhesions, with the always consequent danger of obstruction, may result from any destruction of the serosal surface in which the parietal or visceral peritoneum may adhere to an opposite surface. Without doubt, the greatest cause of intraperitoneal adhesions is the denudation of the peritoneal surface during surgical procedures. Any process, therefore, that cuts down damage to the thin peritoneal layer should result in less inflammatory reaction and fewer adhesions.

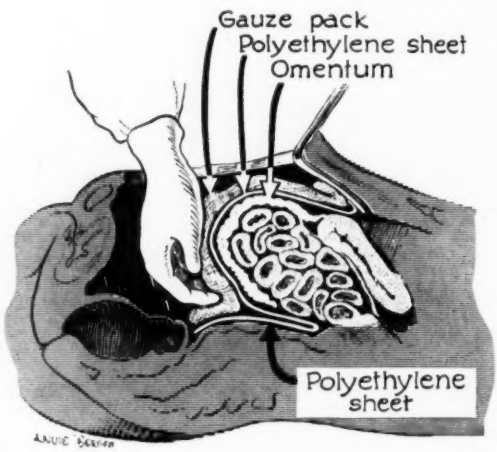

Illustration showing polyethylene sheet placed over all loops of small bowel, which have been first wrapped with omentum.

Customary abdominal packing during abdominal surgical procedures is done with heavy gauze packing. Careful inspection of the bowel after a gauze pack has been against it for a period of time will reveal a dullness of the surface and an impression of the gauze interstices into the serosal wall and a mild inflammatory reaction. In an attempt to prevent this irritation, a new type of abdominal packing has been sought. The material which most ideally fitted this situation is polyethylene [1] film, which is relatively chemically inert and almost entirely free of tissue reaction. When it is moistened with saline, it has a smooth, mildly slippery texture in contrast with the "sandpapery" surface of gauze.

1. Polyethylene film is manufactured by Clay-Adams Co., Inc., New York.

Polyethylene film is available in sheets 54 in. by 0.0015 or 0.002 in. by 10 yd. When divided into sheets 54 in. (137 cm.) wide by approximately 36 in. (91 cm.), it furnishes an ideal material to lay over all peritoneal surfaces, prior to the placing of gauze packs, to protect them from damage. In order to give additional protection to the bowel, the omentum is first wrapped about all loops of the small bowel, and the saline-moistened polyethylene sheet is then placed over these, as illustrated in the Figure. Customary laparotomy packs may then be placed without touching the serosa. Since the polyethylene sheet is waterproof, it likewise has a property of partially protecting the remaining abdominal cavity from spillage.

Polyethylene film may be sterilized in benzalkonium (zephiran®) [2] chloride solution or in a solution of R. I. G.[3] (rust-inhibiting germicide), another quaternary ammonium compound, now called C. R. I. (concentrated rust-inhibiting germicide), for a period of 30 minutes or may be autoclaved in a pack in which towels are placed between the sheets in such a manner that no two surfaces of the polyethylene film will adhere to each other. Autoclaving, however, materially shortens the life of the material and causes the surface to become brittle and cracked after approximately five or six sterilizations. For practical reasons, chemical sterilizations are preferred.

2. Zephiran® is manufactured by Winthrop-Stearns, Inc., New York.

3. R.I.G. is manufactured by Clay-Adams Co., Inc., New York.

HERNIA OF THE LUNG: METHOD OF REPAIR WITH TANTALUM MESH

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VINELAND, N. J.
AND
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BALTIMORE

THE PURPOSE of this paper is to record a successful and satisfactory method of repairing a hernia of the lung.

Hernia of the lung occurs infrequently and not all of those that do occur cause symptoms that require treatment.

Some hernias of the lung, however, are symptomatic, being accompanied by local pain, paroxysmal coughing, hemoptysis, or any combination of the three. In rare instances a lung hernia may become strangulated. A few increase progressively in size until they are large enough to be a nuisance to the patient as well as to cause rejection for employment.

Surgical treatment is indicated for those lung hernias that are symptomatic, strangulated, or progressively increasing in size. As is the case with hernias in other sites, surgery may be elected for those that are asymptomatic as well.

The various surgical procedures used in treating lung hernias date from 1898, when Vogler [1] proposed shifting periosteum or a bone flap from the sternum to cover the defect. In the same year Vulpius [1] bridged a defect by crossing rib strips and securing them with silver wire. This hernia recurred within a year. A cure was reported by Tuffier,[3] who merely freed and ligated the hernial sac. In 1922 Graham [4] gave successful treatment in a case of spontaneous cervical hernia by reducing it and packing the wound with iodoform gauze. In 1935 Winkel [5] effected cure in a

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Cited by Montgomery, J. G., and Lutz, H.: Hernia of the Lung, Ann. Surg. **82**:220-231, 1925.

2. Footnote deleted.

3. Tuffier, cited by Goodman, H. I.: Hernia of the Lung, J. Thoracic Surg. **2**:368-379, 1933.

4. Graham, E. A.: Hernia of Lung and Adenoma of Thyroid, S. Clin. North America **2**:1493-1500, 1922.

5. Winkel, A. H.: Hernia of the Lung: Report of a Case; Outlining Mode of Fascia Lata Repair, J. Thoracic Surg. **4**:627-634, 1935.

case of traumatic intercostal hernia by obliterating the defect with woven strips of fascia lata. Various osteoplastic procedures have been used to close the thoracic defect and prevent recurrence.[6] These procedures, which in the main have been successful, consisted of the following: freeing the sac and closing the parietal pleura, covering as much of the defect as possible with flaps of periosteum reflected from the ribs immediately above and below the defect, and then strengthening the repair by transplanting and anchoring segments of adjacent ribs over the defect. The creation of a moderate pneumothorax "cushion" between the lung and the freshly repaired chest wall was advised by Goodman and by Maurer and Blades. Cases have been reported, however, in which conservative treatment was employed because the defects were considered to be too large for closure by the osteoplastic type of operation.[7]

In 1948 Koontz [8] reported the use of tantalum mesh for repairing hernias of the abdominal wall, and in 1950 Morrow [9] recorded the successful use of the same material in the closure of full-thickness defects in the chest wall.

In the case presented below, closure of the hernial orifice was facilitated by placing a piece of tantalum mesh between the pleura and the ribs adjacent to the defect. The method seemed simpler than those previously described and had the additional advantage that the tantalum repair was strengthened by support from the overlying ribs.

REPORT OF A CASE

C. F. S., a 55-year-old white man, a hospital aide, was admitted to the surgical service of the Perry Point Veterans Administration Hospital on March 19, 1951, complaining of a painful mass which appeared with coughing in the region of a thoracotomy scar.

On Sept. 13, 1950, during a previous hospital admission, the patient had had a symptomatic hiatus hernia repaired. The repair had been made through a left lateral thoracotomy incision, the pleural cavity being entered through the fifth interspace without the removal of rib segments. After an imbrication-type hernioplasty had been performed, the wound was closed in layers with interrupted nonabsorbable surgical (silk) sutures and without the use of pericostal sutures. His postoperative course had been smooth, his wound had healed without difficulty, and the symptoms previously attributed to the hiatus hernia had disappeared.

About one month after operation the patient noticed that with coughing a bulge would appear over the midportion of the operative scar. The mass gradually enlarged until it was about the size of a fist, and the patient then began to experience sharp pain in the area when the mass would protrude. Eventually, he could make the mass appear by contracting the abdominal muscles and closing off the upper respiratory tract. He had had a chronic, nonproductive cough, but there had been no hemoptysis.

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6. Montgomery, J. G., and Lutz, H.: Hernia of the Lung, Ann. Surg. **82**:220-231, 1925. Goodman, H. I.: Hernia of the Lung, J. Thoracic Surg. **2**:368-379, 1933. Maurer, E., and Blades, B.: Hernia of the Lung, ibid. **15**:77-98, 1946. Hurwich, J. J.: Lung Hernia: A Case Report and Review of the Literature, ibid. **18**:261-268, 1949.

7. Pain, A. B.: Hernia of the Lung, Brit. M. J. **1**:59, 1934. Lazar, H.: Traumatic Hernia of the Lung, M. Bull. Vet. Admin. **18**:319-321, 1942.

8. Koontz, A. R.: The Repair of Ventral Hernias with Tantalum Mesh: Preliminary Report, South. M. J. **41**:214-217, 1948; Tantalum Mesh in Hernia Repair: Experimental and Clinical Results, Tr. South. S. A. **60**:394-403, 1948.

9. Morrow, A. G.: The Use of Tantalum Gauze in the Closure of Full-Thickness Defects in the Chest Wall, Surgery **28**:1016-1021, 1950; The Use of Tantalum Gauze in the Closure of a Defect in the Chest Wall, ibid. **29**:288-292, 1951.

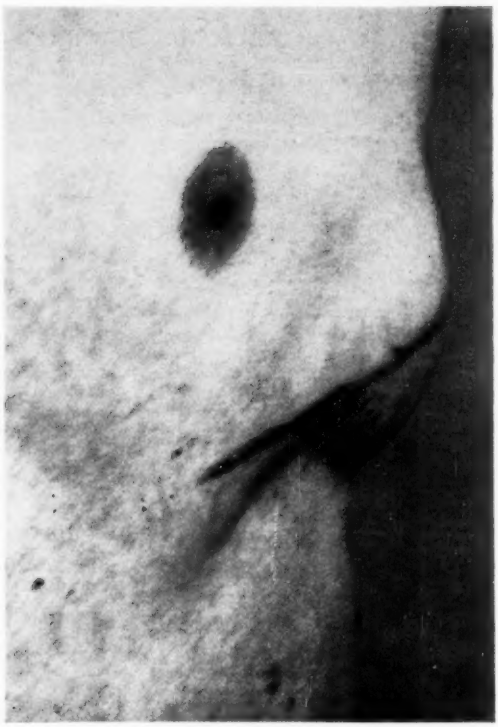

Fig. 1.—Photograph of Patient C. F. S., showing hernia of the lung in the region of a thoracotomy scar during forced expiration.

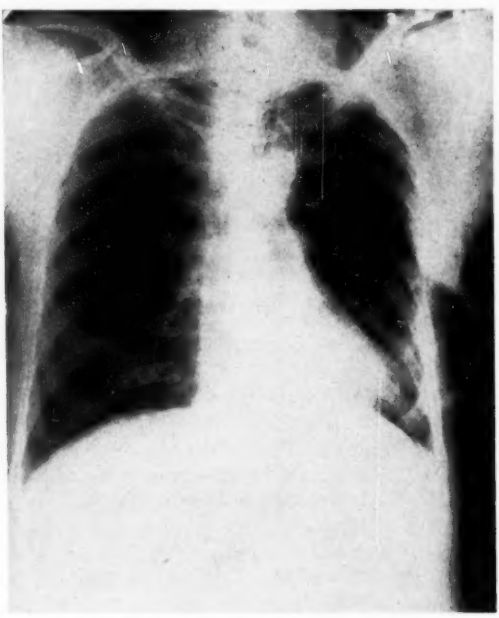

Fig. 2.—Roentgenogram showing herniation of a portion of the left lung during forced expiration. Note separation of fifth and sixth ribs.

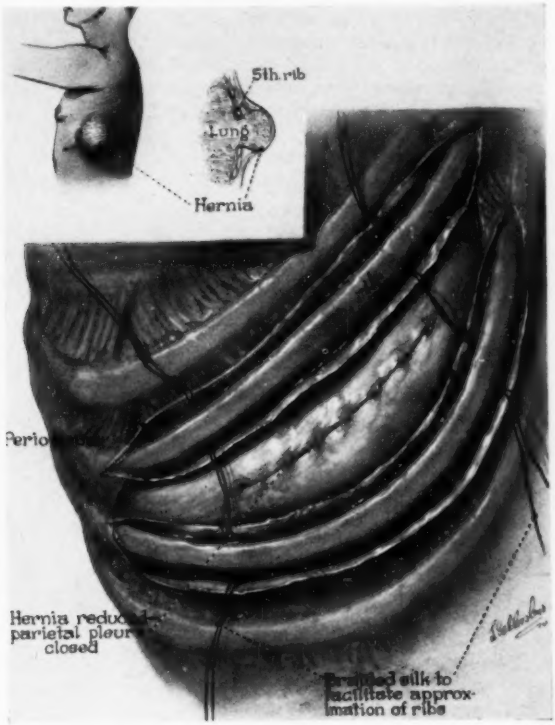

Fig. 3.—Insert illustrates position of hernia and continuity between hernial sac and parietal pleura. Large drawing illustrates method of closing neck of hernial sac (parietal pleura) after redundant portion of sac has been excised.

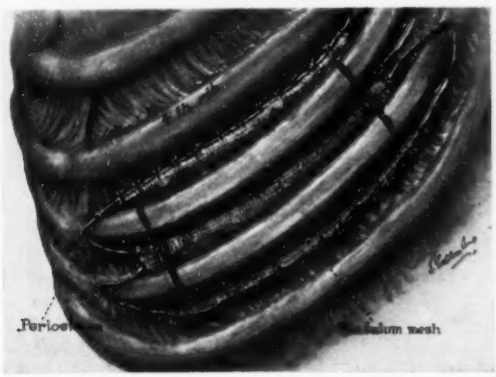

Fig. 4.—Drawing illustrates method of approximating the ribs and inserting tantalum mesh between the pleura and ribs. Each margin of the mesh is folded upon itself so that the tantalum wire sutures used to hold it in place will pass through two layers of the mesh instead of one.

Physical examination on readmission in March, 1951, revealed a well-healed lateral scar overlying and paralleling the fifth interspace on the left. No muscular defect could be palpated, but with forced expiration (Valsalva's maneuver) a mass 6 by 8 by 10 cm. in size appeared deep to the midportion of the scar and seemed to be under considerable tension (Fig. 1). When the abdominal muscles were relaxed or the upper respiratory tract opened, the mass reduced immediately and spontaneously. Roentgenograms taken during forced expiration showed definite herniation of a portion of the lung through a defect in the thoracic cage between the fifth and sixth ribs (Fig. 2).

Since the pain on coughing was becoming severer and the mass was steadily increasing in size, surgical repair seemed indicated. Accordingly, operation was performed on March 20, 1951, with use of general endotracheal anesthesia. The old scar was excised. The silk sutures which had been used for closure of the muscle layers during the previous operation were found to be intact. At the midportion of the incision the fifth and sixth ribs were found to be separated by a distance of almost three fingerbreadths. At the angle of the sixth rib there was a healed

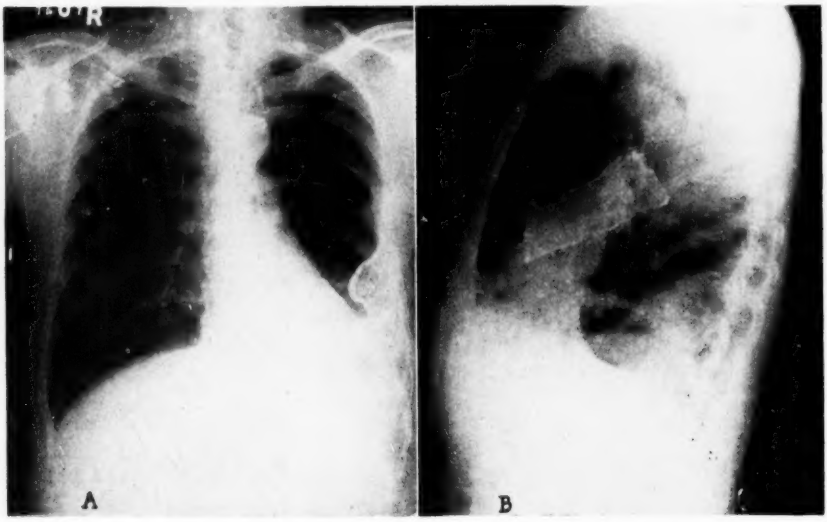

Fig. 5.—Roentgenograms taken nine months after operation show tantalum mesh in its original position and tantalum wire sutures intact.

fracture, the result of gaining exposure during the previous operation. The muscles of the thoracic cage overlying the area where the hernia was known to exist were intact but markedly thinned out. After these muscles had been incised, a large collapsed hernial sac was encountered overlying and adherent to the outer surfaces of the fifth and sixth ribs. When the sac was opened, its interior was found to communicate with the pleural space through a defect between these ribs. The sac itself was continuous with the parietal pleura. The defect between the ribs, occupied by the neck of the sac, was transversely oval and measured about 5 by 14 cm. in size. No lung tissue was adherent to the inner surface of the sac. When intrathoracic pressure was increased by the anesthetist, a portion of the upper lobe of the left lung was seen to herniate through the defect. This portion of lung appeared to be slightly nodular to palpation but was otherwise normal. It was not disturbed. The pleural sac was dissected free from the surrounding muscles and the fifth and sixth ribs. The redundant sac was then excised, and the opening in the parietal pleura was closed with a continuous No. 1 chromic absorbable surgical (gut) suture on an atraumatic® needle. Incisions were then made in the periosteum of the fifth and sixth ribs, and periosteal flaps were developed and reflected downward and upward, respectively. The interval between these denuded ribs was then restored to normal by means of two pericostal

double-strand No. 2 braided nonabsorbable surgical (silk) sutures (Fig. 3). Since the overlying muscles had been found to be so attenuated, and since the hernia was of such magnitude, it was felt that the strength of the repair could be increased substantially by placing a piece of tantalum mesh between the pleura and ribs. Accordingly, a piece of mesh was prepared, each margin being folded upon itself so that the sutures which were to hold it in place would pass through two layers of the mesh instead of one. After the mesh had been placed between the ribs and the pleura it was secured to the periosteum of the fifth rib above and the sixth rib below with interrupted sutures of 0.010-in. (0.254-mm.) diameter monofilament tantalum wire (Fig. 4). By placing the tantalum beneath the ribs, additional support was obtained. The muscles, subcutaneous tissues, and skin were then closed in layers with interrupted silk sutures.

The patient's postoperative course was complicated by a mild febrile rise, which responded satisfactorily to parenteral penicillin therapy. On the seventh postoperative day 75 cc. of serosanguineous fluid were aspirated from a pocket in the deeper layers of the wound. Cultures of this fluid were sterile. Thoracentesis was done on the 11th postoperative day, with recovery of 500 cc. of serosanguineous fluid, cultures of which also showed no growth. During the postoperative period the patient coughed vigorously in spite of sedation but with no evidence of recurrence of the hernia. His wound healed without difficulty, and he was discharged on the 16th postoperative day.

A follow-up examination conducted nine months after operation revealed that the patient was asymptomatic. With forced expiration there was no evidence of recurrence of the hernia. Roentgenograms showed the tantalum mesh in its original position and the tantalum sutures intact (Fig. 5).

SUMMARY

A method of repairing a hernia of the lung with tantalum mesh is presented and its successful use in one case is reported.

REGIONAL DISTRIBUTION OF MOLES AND MELANOMAS

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AND
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THE RELATIONSHIP of pigmented moles and melanomas has been studied with regard to histogenesis, age of subjects, malignant derivation, etc., but the significance of their proportionate regional distribution has been largely ignored. We consider these clinical facts to be of sufficient interest and, perhaps, importance to justify the presentation of this information by four series of scattergrams showing (*a*) the pattern of primary melanin distribution when unaffected by sunlight or other melanin-provoking exposure; (*b*) the distribution of benign pigmented moles in 1,000 white adult patients; (*c*) the distribution of benign pigmented moles in 840 patients who, with their physicians, were so suspicious of these lesions as to have them removed for microscopical examination, and (*d*) the regional distribution of 1,225 verified malignant melanomas.

From an outpatient diagnostic service, 1,000 adult white patients, who came for other complaints, were carefully examined in order to note and record the character, number, and locations of their moles. These patients were unconcerned about moles, so it was assumed that the data so secured could be interpreted as representative of the natural incidence in adult white people. A total of 14,609 nevi were recognized on these 1,000 people, an average of 14.6 moles per subject. Some people have more than a hundred moles and others have only two or three, but we have never examined a mature white adult who did not have at least one discernible mole. It is no exaggeration to state that the mole is the commonest tumor in the entire category of neoplastic diseases. The very frequency of its occurrence lulls suspicion and favors tolerance of these tumors by patients and physicians alike.

The next survey was an attempt to discover how correct was our index of suspicion of moles which worried both patients and physicians sufficiently to have certain specific moles surgically removed. A group of 840 patients had moles removed and microscopically identified as benign: A scattergram shows their regional distribution, which is only significant when compared with the natural distribution of moles in casual subjects and with the locations of malignant melanomas. In general, it may be stated that the percentage distribution of excised moles and malignant melanomas was almost identical for the skin of the trunk and upper extremities. Judging from comparative figures, there was apparently less and insufficient concern for nevi on the lower extremities and a disproportionate eagerness to have moles on the face excised; this last fact, in its true worth, might be due rather to a desire for cosmetic improvement by the removal of these conspicuous lesions than to concern about the hazard of future malignant degeneration.

From the Memorial Cancer Center.

As a matter of interest, we are including a scattergram showing the pattern of primary melanin distribution, when unaffected by sunlight or other melanin-provoking exposure. This chart, based mainly on spectrophotometric data, is furnished through the courtesy of Dr. Edward A. Edwards,[1] of the Department of Anatomy, Harvard Medical School. The scalp was omitted from consideration. The distribution of melanin pigment does not correspond with the regional localization of malignant melanoma, from which fact one might infer that the greater number of melanoblasts at any site does not necessarily predispose the part to the development of melanoma. Melanin disposition in the skin is increased in the regions of great-

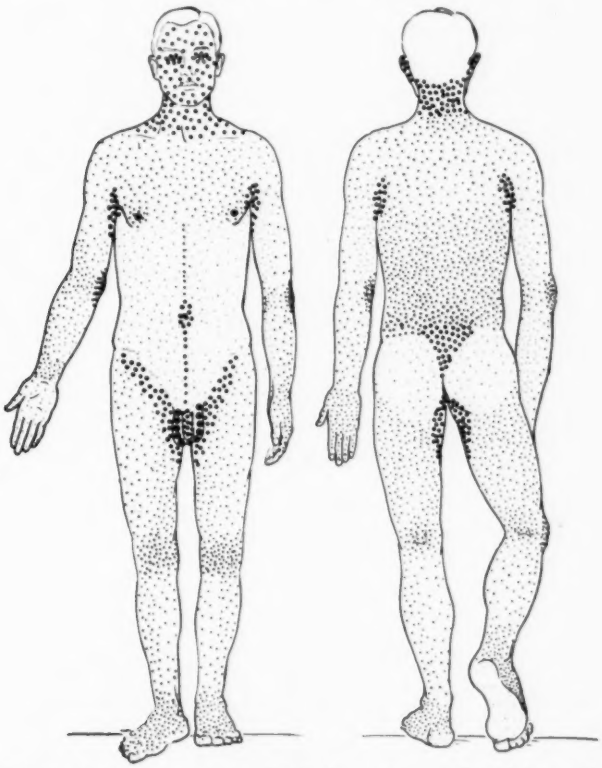

Fig. 1.—Pattern of primary melanin distribution, when unaffected by sunlight or other melanin-provoking exposure, based on spectrophotometric data (courtesy of Dr. Edward A. Edwards, Department of Anatomy, Harvard Medical School).

est concentration of apocrine sweat glands. The greatest discrepancy is found between scarcity of melanin and the frequency of melanoma on the soles.

Nevi are probably all congenital. The term "acquired nevi," read often in dermatologic literature, is probably incorrect and has been based on the observation of nevi appearing for the first time at various periods in postnatal life. The time of origin of nevi and the time of their first visibility are not necessarily the same. Some

1. Edwards, E. A., and Duntley, S. G.: Pigments and Color of Living Human Skin, Am. J. Anat. **65**:1-33, 1939.

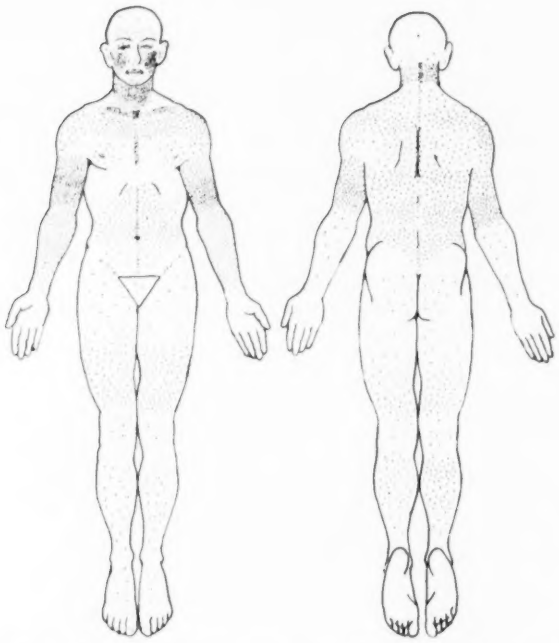

Fig. 2.—Regional distribution of nevi found on general examination, based on 1,000 adult patients. Each dot represents five nevi.

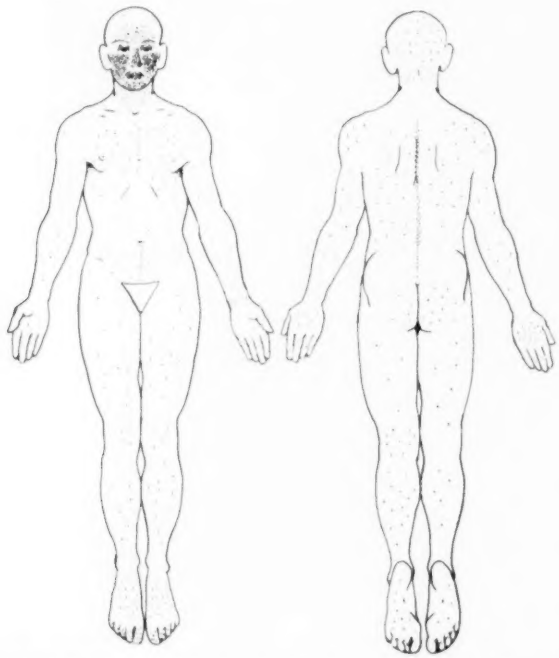

Fig. 3.—Regional distribution of nevi excised at Memorial Hospital, based on 840 patients. Each dot represents one nevus.

moles in newborn infants are invisible at birth but appear after a variable time, perhaps after some exposure has exerted its melanin-provoking influence. Moles acquire a much darker pigmentation during pregnancy, and women may even volunteer the information that "new" moles seemed to have occurred.[2] The range of visibility for the human eye is rather narrow, and many tiny, faintly pigmented moles may not be seen under normal conditions. Some do become visible as pigmented lesions when the skin is examined in a dark room with the aid of Wood's light. This phenomenon may help to explain some unusual facts about malignant

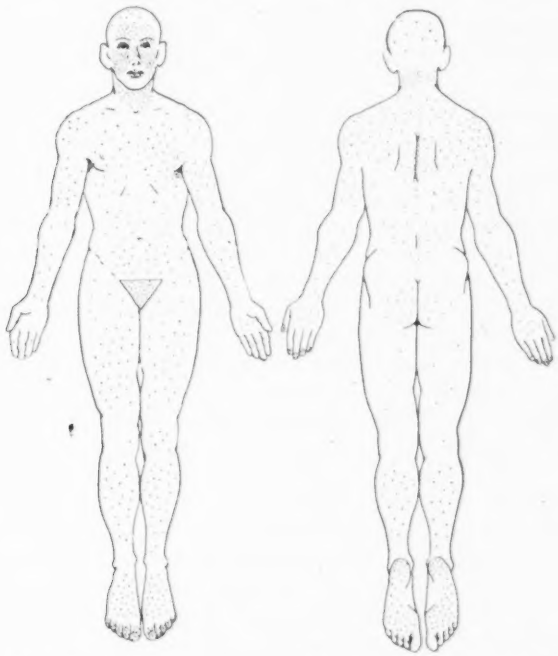

Fig. 4.—Regional distribution of malignant melanomas, based on 1,225 patients. Each dot represents one malignant melanoma. Internal malignant melanomas are not shown above. The distribution of these is as follows: eye 64; oral cavity 18; nasal cavity 8; inner ear 2; lung 1; esophagus 1; anorectal region 19.

melanoma, e. g., the apparent development of malignant melanoma *de novo* at a site which had no precursory skin lesion, according to the opinion of the patient, and the unusual instances in which metastases from malignant melanoma are found and treated, with the site of the primary melanoma remaining cryptic. It may also mean that our scattergram showing the distribution of nevi is incomplete, representing as it does only the visible tumors.

MOLES AND PUBERTY

We have long known of the influence of hormonal stimuli on the behavior of malignant melanoma and its benign precursor, the pigmented nevus. Some deeply

2. Pack, G. T., and Scharnagel, I. M.: Prognosis for Malignant Melanoma in the Pregnant Woman, Cancer **4**:324-334, 1951.

pigmented moles removed in infancy or in childhood have been diagnosed microscopically as true melanomas, as indeed they are, fulfilling all the histological criteria of this malignant tumor, save blood and lymph vessel invasion. In our usage and in this discussion, the term "melanoma" connotes the malignant tumor. In tracing the end-results of treatment of melanomas in infancy and childhood we were long ago impressed by the obvious fact that these children were uniformly cured, often by simple conservative excision, and that the melanoma in subjects this age might be microscopically indistinguishable from its malignant prototype, yet it was clinically benign. In the course of our experience with more than 1,220 verified cases of malignant melanoma, we have never observed a single instance in which a melanoma of infancy and childhood metastasized to nodes and viscera, and we have never witnessed a fatal outcome. There have been rare exceptions to this statement in the experience of others, but our investigations of reputed cases available for our personal study have strengthened our position concerning the rarity of true malignancy in this tumor. We have suggested the term "prepubertal melanoma" for the deeply pigmented tumors that bear the histological earmarks of a malignant melanoma yet do not metastasize or cause death.[3] The distinction here has to do solely with the age, or rather the endocrine status, of the patient. We have on record an instance of true malignant melanoma occurring in an adolescent who had not yet menstruated; this occurrence was not a contradiction of the generalization previously made, because the maiden had developing breasts and axillary and pubic hair, indicating that the hormone factors of puberty were active. Many parents have brought for examination children between the ages of 10 and 14 years in considerable apprehension because of the sudden appearance of numerous dark moles, previously unnoticed or inconspicuous. With approaching pubescence, preexisting moles commonly change to become larger, darker, and more elevated; furthermore, other moles invisible or faintly seen, show increased pigmentary changes. These factors are mentioned only to introduce our thesis, that the pigmented nevus is extraordinarily sensitive to the stimulating influence of these hormones, pituitary, adrenal, and gonadal.[4] We have had the heartbreaking experience of treating too many young girls and boys aged 14 to 20 years for malignant melanomas that could have been prevented by the prophylactic excision of benign precursory nevi in childhood.

RELATIONSHIP OF TRAUMA TO MOLES AND MELANOMAS

The idea has been commonly prevalent for many years that repeated injuries to pigmented moles represent important contributing factors in the conversion of such a mole to a malignant melanoma. In earlier years we held a similar opinion, influenced no doubt by the review of histories of patients who were perhaps prejudiced in their recording of such relationships. The strongest argument in favor of this theory is the great frequency of melanomas occurring on the soles where preexisting moles are subject to the pressure of constant weight bearing. Malignant melanomas are not significantly frequent on the neck and waist, that is to say, along the collar and belt lines where pressure is constantly applied. The trauma of shaving does not appreciably alter the incidence of melanomas on the face, as judged by their proportion of occurrence in males and females. The ordinary mole is not a fragile

3. Pack, G. T.: Prepubertal Melanoma of Skin, Surg., Gynec. & Obst. **86**:374-375, 1948.

4. Pack, G. T.: Management of Pigmented Nevi and Malignant Melanomas, South. M. J. **40**:832-838, 1947.

structure, but the malignant melanoma is, and in consequence of its fragility, soft texture, and vascularity it is easily and repeatedly injured, resulting in early bleeding and ulceration. Reputed instances in which trauma is said to have caused the conversion of a benign nevus into a malignant melanoma, on critical inquiry leave considerable doubt as to whether the tumor originally was a nevus or an early melanoma. We are inclined to the viewpoint that the histological character of the mole, for example a junctional nevus, is of infinitely greater importance in determining the possible conversion to the malignant tumor than is the accident of injury.

NEVI AND MELANOMAS OF THE EYE

Melanoma of the conjunctiva is rare but does occur with about the same frequency as benign pigmented nevi in the same structure. We have seen congenital pigmented moles occurring on the conjunctivae, more commonly near the internal or external canthus of the eye, and developmental anomalies suggesting a primitive nictitating membrane. Except by comparison of character and rate of growth of our microscopic study over some time it is extremely difficult to establish a differential diagnosis between some benign pigmented nevi of the uveal tract and malignant melanomas.

NEVI AND MELANOMAS OF THE HEAD AND NECK

Only 13.2% of moles, as they occur in the general population, are situated in the skin of the head and neck, but 28.9% of malignant melanomas are situated in this same regional distribution, indicating that the malignant tumor occurs twice as frequently as the benign tumor in a relative instance. Of the benign nevi excised, 49.4% were on the face and neck, which indicates that the purpose for their removal was more for cosmetic reasons than for fear of the hazard of malignant degeneration. The 31 malignant melanomas occurring in the oronasal cavity are only 2.5% of the entire number seen and treated. Pigmented nevi that can be identified as such are of extraordinary rarity in the mucosa of the mouth, oral cavity, and nasal accessory sinuses. The inability to detect the precursory lesions accounts for the failure to practice prophylactic excision and perhaps accounts in part for the low rate of curability of melanomas in this location, as they are almost inevitably fatal. Even though nevi are not ordinarily seen or recognized, melanoblasts are present, as was demonstrated by George F. Laidlaw,[5] with use of selective silver staining. For example, he found melanoblasts to occur with considerable frequency on the gums.

GENITAL MOLES AND MELANOMAS

Of a group of 1,000 patients inspected for the detection of moles, with a total of 14,609 nevi in their skin, only 14 such moles were discovered on the skin of the genitals in both the male and the female. The regional distribution comprised only 0.1% of all moles, as compared with the relative frequency of melanomas, of which 35, or 2.8%, of 1,222 melanomas were situated in the skin of the male and the female genitals. Two conclusions may be drawn from these figures, namely, that the malignant melanoma is proportionately twice as common as its benign prototype, the pigmented nevus, and secondly, that due to the relative infrequency of moles on the genital skin, one might conclude that they were more often predisposed to undergo malignant degeneration than moles in other locations. A natural

5. Laidlaw, G. F., and Cahn, L. R.: Melanoblasts of the Gum, J. Dent. Res. **12**:534-537, 1932.

corollary to this would be the wisdom of routine excision of all moles involving the genital skin as a proper prophylactic measure for the prevention of genital melanomas.

ANORECTAL MELANOMAS

Considering the thousands of proctoscopic examinations which are done every year, it is surprising that pigmented nevi are not commonly found in the anorectal mucosa. It is indeed seldom that any are excised and studied microscopically for confirmation. This is unfortunate, inasmuch as it removes all opportunity for the prevention of anorectal melanomas, which, in most cases, have been of fatal termina-

TABLE 1.—*Location of Malignant Melanomas, of Nevi Excised, and of Nevi Found on General Physical Examination*

Location	Malignant Melanomas		Nevi Excised		Nevi Found on General Physical Examination	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
Number of patients	1,222		840		1,000	
Number of lesions	1,235*	100.0	1,070†	100.0	14,609‡	100.0
Head and neck (total)	357	28.9	529	49.4	1,922	13.2
Skin	263	21.3	529	49.4	1,922	13.2
Oronasal	31	2.5	0	0.0	0	0.0
Eye	63	5.1	0	0.0	0	0.0
Upper extremities (total)	135	10.9	97	9.1	4,419	30.2
Upper arm	45	3.6	26	2.4	2,906	19.7
Forearm	44	3.6	33	3.1	1,513	10.4
Dorsum of hand	16	1.3	38	3.6	0	0.0
Palm	13	1.1	0	0.0	0	0.0
Subungual	17	1.3	0	0.0	0	0.0
Trunk (total)	291	23.6	269	25.1	5,746	39.3
Chest	74	6.0	82	7.7	1,256	8.6
Abdominal wall	47	3.8	66	6.1	1,000	7.5
Shoulder	68	5.5	24	2.2	845	5.8
Upper back	70	5.7	51	4.8	1,313	9.0
Lower back	32	2.6	46	4.3	1,232	8.4
Lower extremities (total)	366	29.6	175	16.4	2,508	17.2
Upper leg and buttocks	99	8.0	56	5.2	1,597	10.9
Lower leg	88	7.1	50	4.7	857	5.9
Dorsum of foot	100	8.1	58	5.4	49	0.3
Sole	59	4.8	11	1.0	5	...
Subungual	20	1.6	0	0.0	0	0.0
Genitalia	35	2.8	0	0.0	14	...
Anorectal	19	1.5	0	0.0	0	0.0
Primary site not determined	32	2.6	0	0.0	0	0.0

* In 13 instances there were primary multiple melanomas.
† There were 140 cases with multiple lesions which were excised, not including the multiple lesions not excised.
‡ The average number of nevi per patient was 14.6.

tion. Of 1,222 cases of malignant melanoma in this series, 19, or 1.5%, were located in the anorectal mucosa. Melanoblasts are of some frequency in mucocutaneous junctions but seldom participate in the formation of congenital pigmented nevi.

MELANOMAS OF THE EXTREMITIES

The fact which we had long recognized clinically but which had only recently had statistical proof was the disproportion of frequency of malignant melanomas on the lower extremity as compared with the upper extremity. From the study of Table 1 it will be seen that 30.2% of moles were situated in the skin of the upper extremities, whereas only 10.9% of malignant melanomas were so located. Per contra, only 17.2% of the pigmented moles were in the skin of the lower extremities, whereas 29.6% of melanomas were in the skin of the lower extremities. Stated

in another way, moles occur almost twice as frequently in the upper extremities as compared with the lower extremities, and melanomas occur almost three times as frequently on the lower extremity as compared with the upper extremity. Visible pigmented moles are infrequently encountered on the soles; only 5 such moles were identified on the 2,000 soles of the 1,000 patients, or a total of 5 of 14,609 moles generally distributed on these patients. If these figures are of any significance, a wise precaution would be the routine removal of all pigmented nevi found on the soles. This admonition we offer is constantly practiced by us.

Selective silver staining has shown that melanoblasts or melanotic whitlows are not infrequent, comprising 37, or 2.9%, of the malignant melanomas in this series. Pigmented nevi are not so frequently encountered in the nail bed and, when seen, should be surgically excised in their entirety in order to be available for microscopic study; in our experience, the melanoma occurs much more frequently in the nail bed than does the pigmented nevus. The advice to perform routine excision and

TABLE 2.—*Nevi of the Hands and Feet* *

Type of Nevi	Hands		Feet	
	No.	%	No.	%
Total nevi	32	100.0	42	100.0
Junctional nevi (dermoepidermal or marginal nevi)	2	6.2	3	7.1
Intradermal nevi (common moles or neuronevi)	3	9.4	5†	11.9
Blue nevi (Jadassohn-Tieche nevi)	18	56.3	4	9.5
Compound nevi	9	28.1	27‡	64.3
Prepubertal melanoma	..	....	3	7.1

* A group of nevi on the hands and feet were surgically excised. Dr. Sophie Spitz reviewed these tumors, according to the classification by Arthur C. Allen [6] which we employ at the Memorial Cancer Center. (From monograph in preparation by Robert Booher and George Paul.)
† One patient had two lesions.
‡ Three patients had two lesions.

microscopical study of these pigmented lesions of the nail bed is based on the clinical difficulty in establishing the differential diagnosis between a benign lesion and a malignant lesion.

MELANOMAS OF UNDETERMINED PRIMARY SITE

One of the most baffling problems in the management of malignant melanomas has been the occasional instance in which metastatic melanoma is discovered, with no discernible evidence of a site of the primary tumor. Our policy has been to treat the melanoma wherever it is found. In these circumstances this treatment is performed as a palliative measure, with little expectation of cure. There has been one instance in our experience in which a 17-year cure resulted from an axillary dissection of metastatic melanoma; the patient has been constantly observed in the 17-year interval, with no recurrent activity of the cryptic primary melanoma. This is the only definitive cure out of 32 cases (2.6% of the entire series) of malignant melanomas of undetermined primary source in our group.

REPORT OF A CASE

Metastatic Melanoma in Axillary Lymph Node Without Discernible Primary Lesion, 17-Year Definitive Cure.—M. P., a 54-year-old man, was first seen on July 12, 1935. For five months he had been aware of a slowly enlarging lump in the right armpit. On June 19, this lump was surgically excised and proved to be a large axillary lymph node containing metastatic melanoma.

6. Allen, A. C.: Reorientation on Histogenesis and Clinical Significance of Cutaneous Nevi and Melanomas, Cancer **2**:28-56, 1949.

His past history was unimportant. He had been treated for duodenal ulcer intermittently during the previous 15 years. Repeated questioning failed to secure any information concerning the previous removal of any similar lesion in the skin. According to the patient's knowledge, no pigmented lesion had ever been electrodesiccated or surgically excised or had disappeared spontaneously.

Physical examination revealed a normal adult man, with no suspicious nevi on the corresponding arm, shoulder, or trunk. Examination of his eyes by the ophthalmologist, Dr. Algernon Reese, revealed no suspicious foci of pigmentation within the uveal tract or in the fundus.

Operation.—The patient was admitted to the Memorial Hospital for Cancer and Allied Diseases where a radical axillary dissection was performed. All the remaining lymph nodes were examined microscopically; none were found to contain melanoma. A roentgenogram of the chest revealed no evidences of pulmonary metastasis.

Course.—This patient was examined at frequent intervals during the succeeding 17 years, and at no time had the primary melanoma become visible, nor had any other evidence of the disease been manifest. The result was a 17-year survival-period following the removal of a lymph node in the right axilla, which contained metastatic melanoma, as diagnosed by Dr. Fred W. Stewart, pathologist to the Memorial Hospital.

TABLE 3.—*Five-Year End-Results in the Treatment of Melanoma According to Location (1917-1945)*

Location	Total Cases	Determinate Cases †	Five-Year Definitive Cures	
			No.	%
Total cases	744	575	123	21.4
Skin of the head and neck	173	128	23	18.0
Eye	47	35	9	25.7
Oronasal	17	11	1*	9.1
Arm	58	46	8	17.4
Palm	11	8	7	87.5
Subungual	24	18	7	38.9
Leg	125	98	30	30.6
Sole	59	48	15	31.2
Trunk	162	131	18	13.7
Genitalia	31	25	4	16.0
Anorectal	16	10	0	0.0
Primary site not determined	21	17	1	5.9

* In the sixth postoperative year, pulmonary metastases developed.

† Determinate cases exclude those who refuse treatment, discontinue treatment or are lost trace of in less than five years from admission.

END-RESULTS IN THE TREATMENT OF MELANOMA ACCORDING TO REGIONAL DISTRIBUTION

The percentage of definitive cures of malignant melanoma bears an important relationship to the area of skin involved with the primary tumor as well as to the stage of the disease.[7] In Table 3 are recorded the definitive cures of all patients treated through 1945, based on the site of the primary lesion. The prognosis is seen to be uniformly bad for melanomas of oronasal and anorectal distributions.

Considering the high grade of malignancy of melanoma, the frequency of metastases, and the low rate of curability (21.4%) as a whole, the prognosis for melanomas occurring primarily in the nail bed, leg, and sole is surprisingly good. The extraordinary phenomenon of seven out of eight patients with palmar melanomas falling into the five-year definitive-cure group can perhaps be explained by the conspicuous location of melanomas in the palm, serving as a constant reminder of its presence and leading to early detection and immediate removal.

7. Pack, G. T.: The Problem of Malignant Melanoma, Cancer, to be published. Pack, G. T.; Gerber, D. M., and Scharnagel, I. M.: End-Results in Treatment of Malignant Melanoma, Ann. Surg. **136**:905-911, 1952.

ION-EXCHANGE RESINS AS ADJUNCTS IN TREATMENT OF HIGH INTESTINAL FISTULAE

Clinical and Experimental Study

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THE PURPOSE of this paper is to report our experiences in the use of ion-exchange resins in the treatment of high intestinal fistulae and the results of some experimental work on the antitryptic activity of these substances. Because polyamine-formaldehyde anion-exchange resins have been reported to have an inhibitory effect on tryptic enzyme activity,[1] it occurred to us that these resins might be of use in reducing the cutaneous excoriation accompanying gastric, duodenal, and high jejunal fistulae.

INTRODUCTION

In 1945 Segal [2] first suggested the use of anion-exchange resins in the treatment of peptic ulcer. He found that these substances reduced gastric acidity by absorbing free hydrochloric acid and mitigating the activity of the tryptic enzymes of the stomach by altering the pH of the medium. Martin and Wilkinson [1] later found that anion-exchange resins deactivated 80% of the gastric pepsin when they were mixed with a solution of gastric pepsin and N/10 HCl. They also stated that gastric trypsin lost 64% of its activity when mixed for 15 minutes with an anion-exchange resin. Segal, on the other hand, denied the specificity of resin antitryptic activity and showed that such action was only secondary to a pH change.[2]

During the past five years, anion-exchange resins have been used with benefit in the treatment of peptic ulcer.[3] The resins have been shown to be almost completely nontoxic, even when given in large amounts by mouth.[2]

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1. Martin, G. J., and Wilkinson, J.: The Neutralization of Gastric Acidity with Anion Exchange Resins, Gastroenterology **6**:315, 1946.

2. Segal, H. L.; Hodge, H. C.; Watson, J. S., Jr., and Scott, W. J. M.: Polyamine-Formaldehyde Resin: Its Effect upon the *p*H of Acidified Solutions and the *p*H and Pepsin of Gastric Juice in Vitro; Its Toxicity in Rats; Preliminary Feeding Tests, Gastroenterology **4**:484, 1945.

3. Spears, M. M., and Pfeiffer, M. C. J.: Anion Exchange Resin and Peptic Ulcer Pain, Gastroenterology **8**:191, 1947. Kraemer, M.: Antacid Therapy for Peptic Ulcer, Connecticut M. J. **12**:305, 1948. Kraemer, M., and Lehman, D. J., Jr.: The Treatment of Peptic Ulcer with Anion Exchange Resins, Gastroenterology **8**:202, 1947.

CLINICAL RESULTS

A series of patients having duodenal and high intestinal fistulae were given polyamine-methylene resin (resinat [4]) both orally and locally applied to the fistulous opening. The material used in this study was of 200 mesh, this being the particle size used clinically in the management of peptic ulcer and apparently best suited for antitryptic and alkalinizing effects. The material is a finely granular water-insoluble amber-colored powder.

REPORT OF CASES

Case 1 (St. Louis City Hospital).—In M. J. R., a 50-year-old white woman, a duodenal fistula developed after the performance of a cholecystectomy, duodenotomy, and sphincterotomy for chronic pancreatitis. By the fourth day the skin was excoriated, and as much as 1,125 ml. of fluid was obtained from the fistula opening by suction drainage. The instillation of 1 gm. of polyamine-methylene resin in 15 ml. of water every hour through a gastric tube was begun on the sixth day, after all other measures had failed to control the excoriation. These measures included continuous gastric suction and complete parenteral alimentation, as well as the application of various pastes and chemicals to the fistulous opening. Drainage through the fistula decreased within 12 hours and after 48 hours was dry and remained closed.

Case 2 (Barnes Hospital).—In E. C. B., a 33-year-old white woman, a duodenal fistula developed on the second postoperative day following reconstruction of the common bile duct. By the fifth postoperative day the fistulous drainage was still large, and severe excoriation of the skin of the anterior abdominal wall had developed. Polyamine-methylene resin was applied locally and given by mouth (1 gm. every hour in 15 ml. of water). The local application of polyamine-methylene resin was followed almost instantly by relief from pain. Within 72 hours the fistula was completely closed, and the skin quickly healed.

Case 3 (Barnes Hospital).—S. G., a 40-year-old man, had a duodenal fistula following his seventh laparotomy, with the diagnosis of regional enteritis. Five unsuccessful attempts were made to close the duodenal fistula during a 16-day period. After the fifth attempt at operative closure, polyamine-methylene resin was sprinkled on the excoriated skin, and 1 gm. was given orally every hour. The resin relieved the stinging pain around the duodenal opening, and within three days the wound had filled in remarkably. By the end of seven days the duodenal fistula was closed. Jejunal and large bowel fistulae that were also present during this period remained open and were apparently unaffected by oral or local therapy with polyamine-methylene resin.

A number of lower small bowel fistulae have been studied, but in them no benefit has been noted from the use of the resin either orally or locally.

These cases and three others with a similar clinical course formed a rather suggestive mass of evidence that anion-exchange resins were effective when given by mouth and when applied locally in controlling the digestion of the wound of high intestinal fistulae. One cannot necessarily conclude that the resins accelerated the closure of the high intestinal fistulae, since many such lesions spontaneously close within 3 to 80 days after their appearance. Their clinical course is often extremely unpredictable.

Because of the variability of the clinical course of patients with intestinal fistulae, it was decided to test the antitryptic activity of ion-exchange resins in vitro against gastric and duodenal juice.

4. The "resinat" was furnished by Mr. Gustav J. Martin, of the National Drug Company, Philadelphia.

EXPERIMENTAL METHODS AND RESULTS

The tryptic activity of gastric and duodenal juice was determined by the method of Anderson,[5] using gelatin as a substrate and detecting enzyme activity by the minimal concentration of intestinal juice causing liquefaction. Gastric and duodenal juice was obtained from intubated hospitalized patients.

A set of 10 test tubes was filled with a given amount of polyamine-methylene resin. The first set contained 10 mg. of polyamine-methylene resin, and corresponding sets of tubes contained up to 400 mg. of the resin. A control set of tubes into which no polyamine-methylene resin had been added was run on each sample of intestinal juice. Each group of tubes was then filled with increasing concentrations of intestinal juice, diluted so that 1 cc. was equivalent to a minimum of 0.00125 and a maximum of 0.32 cc. of undiluted intestinal juice. In this way each concentration of intestinal juice was tested for tryptic activity against increasing concentrations of polyamine-methylene resin.

Since pH change was of importance in this type of study, isotonic sodium chloride solution was used in place of the $NaHCO_3$ buffer, as described by Anderson.[5] This allowed adequate dilution without any pH change.

The suspension of polyamine-methylene resin in varying concentrations of intestinal juice was then placed on a mechanical shaker for 30 minutes. It was appreciated that all tubes in any one study should be exposed to the same amount of shaking, for, as has been shown,[6] one factor in ion-exchange activity is the surface area of resin exposed to the ions involved, which in turn depends on particle size and amount of agitation. After this suitable mixing period, the pH was determined and recorded for each sample.

To each sample 2 cc. of a liquefied 7.5% gelatin solution warmed in a 37 C. incubator was then added and the mixture again stirred by inverting each tube three times. The resin intestinal juice suspension, together with the gelatin substrate, was then incubated for one hour in a 37 C. water bath. The tubes containing this liquid were then placed in the cold room overnight at a temperature of 0 C. The next morning (12 to 20 hours later) the tubes were removed and read.

Tryptic activity was measured by the minimal concentration of intestinal juice that resulted in liquefaction of the gelatin substrate. Such an end-point was determined by inversion of the tubes.

Five samples of gastric juice were tested in this manner against varying concentrations of polyamine-methylene resin and intestinal juice.

The Chart shows a typical response of tryptic activity to increasing amounts of polyamine-methylene resin. It will be noted that as the amount of exchange resin increases, tryptic activity decreases. Simultaneous pH determinations upon these same tubes indicate that such increasing amounts of anion-exchange resin progressively decrease the acidity of the medium and therefore antagonize the action of the acid pepsin of the stomach.

In order to demonstrate that such antitryptic activity was entirely the result of pH changes, the reversibility of such enzyme antagonism was effected by lowering

5. Anderson, D. H.: Pancreatic Enzymes in the Duodenal Juice in the Celiac Syndrome, Am. J. Dis. Child. **63**:891, 1942.

6. Martin.[1] Segal.[2]

the pH. In each of the specimens of gastric juice a tube was made with 100 mg. polyamine-methylene resin and 0.01 cc. of gastric juice. This was prepared with gelatin, in the manner described above, and incubated. After cooling, the pH of these samples was between 5 and 6. None showed tryptic activity, as tested by gelatin liquefaction. Hydrochloric acid N/10 was then added dropwise until a pH of 2 was reached. The tubes were then again thoroughly shaken for 30 minutes, reincubated, and cooled as before. In each case the tryptic activity was present at such an acid pH despite the presence of the exchange resin in a relatively high concentration.

Duodenal and small intestinal tryptic enzyme activity was similarly tested with anion-exchange resin. In no case was the alkalinity of the succus entericus altered, and in no case was the tryptic activity of such juice changed.

In two clinical cases, duodenal and high intestinal juice was collected from fistula openings both before and after the administration of polyamine-methylene resin by mouth, but no change in tryptic activity could be demonstrated.

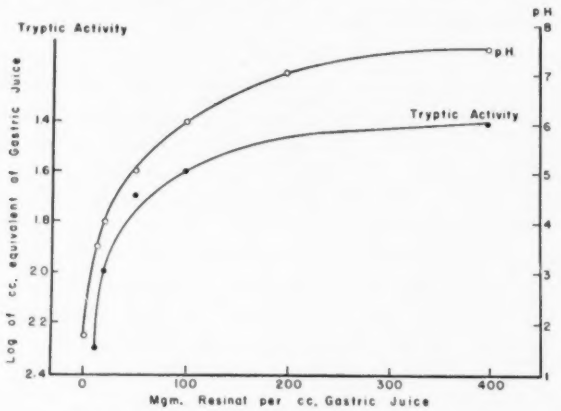

Chart shows a typical response of tryptic activity to increasing amounts of polyamine-methylene resin (resinat).

COMMENT

This experimental evidence suggests that any antitryptic enzyme effect inherent in the anion-exchange resin under study is wholly dependent upon the alteration of acidity of the medium and not in a specific inhibitory manner. Gastric (acid) trypsin is made less effective as neutrality and alkalinity is approached. The activity of alkaline trypsin, as found in the duodenum and small intestine, is unaltered by polyamine-methylene resin.

A series of duodenal fistulae has been presented which have closed during the course of oral and local therapy with polyamine-methylene resin. Experimentally, this agent has been shown to have no effect upon alkaline trypsin. One therefore wonders whether acid gastric trypsin does not play a part in keeping duodenal fistulae open and whether it does not contribute significantly to digestion of the surrounding skin and granulation tissue. It is conceivable that rushes of acid gastric juice might pour through such fistulae. Should this be the case, anion-exchange

resins, by virtue of their amphoteric properties, might aid in closure of such fistulae. Such seemed to be the situation in the cases herein reported.

It is recognized that duodenal fistulae are notoriously unpredictable in their tendency toward closure, and such speculation is only hypothetical.

The clinical management of acute high intestinal fistula remains an unsolved problem. The wide variety of operative, mechanical, and chemical devices proposed in the past to aid in the closure of acute intestinal fistula are occasionally of benefit but are by no means specific. The problem, it would seem, is to find some substance that can be given by mouth with impunity that temporarily, at least, will inhibit tryptic activity, thus allowing natural reparative processes to close the involved segment of intestine, unhindered by constant digestion from proteolytic enzymes.

SUMMARY

An anion-exchange resin, polyamine-methylene resin (resinat), widely used in the treatment of peptic ulcer, has been tried as an adjunct in the closure of high intestinal fistulae because of its alleged antitryptic activity.

Six patients with duodenal fistulae have been treated with an anion-exchange resin (polyamine-methylene resin). The material was given by mouth and applied locally. Spontaneous closure of these fistulae occurred in all cases between the 5th and 10th day following drug administration. Three of these cases are reported in some detail.

Because of the extremely varied clinical course of high intestinal fistulae, no conclusion can be drawn as to the effectiveness of polyamine-methylene resin in aiding the closure of high intestinal fistula. The excoriation of the skin about the fistula quickly subsided after the administration of polyamine-methylene resin.

The antitryptic activity of the anion-exchange resin experimentally tested was limited to its effectiveness in changing the pH of the intestinal contents to a point at which the enzyme was ineffective. The proteolytic activity of gastric acid pepsin was reduced by the acid buffering capacity of the resin and not by any specific antitryptic effect thereof. The activity of duodenal pepsin was unaltered by the resin studied.

It is likely that anion-exchange resins are no more effective in controlling excoriation and aiding in closure of high intestinal fistulae than other buffers previously given ample clinical trial.

STERNUM-SPLITTING INCISION FOR UPPER ABDOMINAL SURGERY

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STERNOTOMY has been used by a number of people, including Blalock [1] and Clagett [2] for thymectomy and Holman [3] for pericardiectomy. As far as is known, however, Wangensteen [4] is the first to have used it for upper abdominal surgery. Combined with a midline supraumbilical incision, Wangensteen states that it has come to be the incision of choice at the University of Minnesota Clinic in operations on the stomach, liver, diaphragm, and spleen. In the cases in which I have used it, it has afforded unusually good exposure with a very benign postoperative course.

TECHNIQUE

A midline incision is made from the xiphoid to just above the umbilicus and the peritoneal cavity entered. Thorough exploration is performed, and it is determined whether the addition of sternotomy is necessary to perform the surgical procedure at hand. If so, a small incision is made in the tissues just beneath the xiphoid and the finger inserted into the anterior mediastinum just under the sternum. Care is taken not to enter either pleural cavity. If available, a Lebsche sternum knife is used to split the sternum to the desired level. Otherwise a chisel and hammer may be used with a small ribbon retractor beneath the sternum. Splitting is performed usually to the level of the fourth or fifth intercostal space. The incision may or may not be carried diagonally across the sternum to the intercostal space. A short Finochietto retractor is then inserted and the sternum gradually spread. The diaphragm is incised between successive Kelly snaps as far as desired. For certain procedures, it is only necessary to divide the diaphragm a short distance. For others, it affords more exposure to divide the diaphragm to and through the esophageal hiatus. Suture ligatures are placed through the diaphragm beneath each snap. It is usually easy to separate the pericardium from the diaphragm. In recurrent explorations there is occasional difficulty in this procedure, but no unusual

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From the Department of Surgery and the Third Surgical Service, Boston University School of Medicine, and the Boston City Hospital.

1. Blalock, A.; Mason, M. F.; Morgan, H. J., and Riven, S. S.: Myasthenia Gravis and Tumors of the Thymic Region, Ann. Surg. **110**:544-561, 1936.

2. Clagett, O. T., and Eaton, L. M.: Surgical Treatment of Myasthenia Gravis, J. Thoracic Surg. **16**:62-80, 1947.

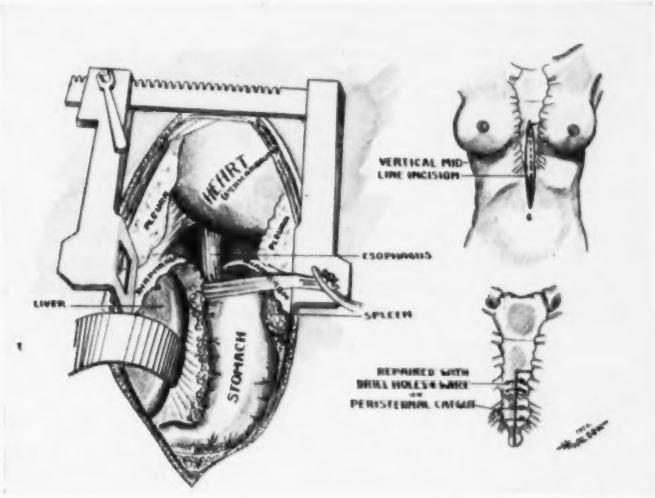
3. Holman, E., and Willett, F.: The Surgical Correction of Constrictive Pericarditis, Surg., Gynec. & Obst. **89**:129-144, 1949.

4. Wangensteen, O. H.: Cancer of the Esophagus and Stomach, New York, American Cancer Society, Inc., 1951; Segmental Gastric Resection for Peptic Ulcer, J. A. M. A. **149**:18-23, 1952.

trouble has been encountered. When the surgical procedure is completed, the diaphragm is joined with interrupted nonabsorbable surgical (silk) sutures. The esophageal hiatus is adjusted to the proper size. The sternum may be approximated either by drilling holes and passing wire through and tying the wire together or by the use of peristernal sutures of surgical gut. The abdominal wall is usually closed with interrupted figure-of-eight near-and-far sutures of braided wire to all layers except the skin.

REPORT OF CASES

CASE 1.—W. R., aged 44 years, had a perforated duodenal ulcer in 1940 and a partial gastrectomy in 1947. He felt well for about four years and then began to have episodes of pain and vomiting, occasionally with blood and tarry stools. On Jan. 30, 1952, exploration was performed. The previous resection appeared adequate, and a diagnosis of marginal ulcer was made. Because of the adhesions in the upper abdomen and because the costal angle was acute and the esophageal



Large figure on left represents exposure for gastroesophageal resection or for repair of hiatal hernia.

hiatus at a high level, the sternum was split up to the level of the fifth interspace and the diaphragm divided for a distance of 2 in. (5.08 cm.). It was then possible to expose the vagi very easily and to resect a portion of each. Holes were bored in the sternum and the sternum closed with two sutures of fine braided wire. The abdominal wall was closed with interrupted near-and-far braided wire sutures to all layers except skin. The postoperative course was very smooth. There was slightly more than the usual amount of postoperative pain for the first few days, but the ordinary amounts of medication were adequate to relieve him. He left the hospital on the eighth postoperative day. A short time later he returned to his occupation as a painter and has been working without difficulty since.

CASE 2.—E. L., a 46-year-old white man, was treated for an extensive carcinoma of the stomach with metastases. He presented a seven-month history of epigastric distress, progressive anorexia, a 50 lb. (22 kg.) weight loss, and increasing dysphagia. On Jan. 26, an operation was performed through a median supraumbilical and sternum-splitting incision. Extensive infiltrating carcinoma was found involving the cardia and many nodes. A gastroesophagectomy was performed, anastomosing the antrum to the esophagus. The spleen was removed with the specimen. The line of transection was about 5 cm. above the cardia, although the specimen itself

showed only about 2 cm. of esophagus. A two-layer fine silk anastomosis was performed. The diaphragm was closed with interrupted silk sutures and the sternum with pericostal absorbable surgical (gut) sutures. On the third postoperative day, sharp epigastric pain occurred. Conservative treatment was employed, and the pain subsided. Administration of oral fluids was begun on the 8th postoperative day, and the patient left the hospital on the 18th postoperative day. In the early postoperative period, moderate discomfort in the region of the sternum was present, but it disappeared before the patient left the hospital.

CASE 3.[5]—A. M. L., a 55-year-old, white, married woman, had a transthoracic repair of a diaphragmatic hernia in December, 1948, which had caused symptoms for about 20 years previously that were not relieved with medical therapy. After the operation, she had a good deal of pain in the incision, and this discomfort persisted intermittently up to the present. About four months postoperatively the symptoms recurred and slowly became worse until April, 1951, when repair was performed. A moderate-sized recurrent diaphragmatic hernia was found. There was considerable scarring around the hiatus and between the pericardium and diaphragm. A median supraumbilical and sternotomy incision was made; the sternum was spread and the diaphragm cut to and through the hiatus. The right pleural cavity was inadvertently entered. The gastric reflection of the sac was excised, the esophagus freed, and the diaphragmatic crura sutured together behind the esophagus. The hiatus and diaphragm were then reconstructed. The pleural hole was not closed. The sternum was approximated with peristernal gut sutures and the abdominal wall closed with interrupted near-and-far braided wire sutures to all layers except the skin. The patient volunteered the information that her postoperative course was much more benign than that following the thoracotomy.

COMMENT

The use of the sternum-splitting incision has given excellent exposure. The heart may be in the way when it is felt desirable to draw down a large amount of esophagus. As seen in Case 2, when there is a question of level of resection, with the possibility that higher esophageal resection may be necessary, it may be wiser to use the transthoracic approach. The lower portion of the incision may be carried to either side of the umbilicus if needed. Because of the possibility of entering the pleural cavity, endotracheal anesthesia should be used.

5. Dr. S. Armstrong assisted in Case 3.

INCIDENCE OF UNSUSPECTED CARCINOMA IN THYROID DISEASE OCCURRING IN A NONENDEMIC AREA

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AND
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BECAUSE there appears to be disagreement on the question of how frequently carcinoma occurs in nontoxic nodular goiter,[1] it seemed pertinent to analyze our experience with such cases occurring in an area in which goiter is not endemic.

This report is based upon an analysis of 200 consecutive cases of thyroid disease in which operation was performed at the Wadsworth Hospital, Veterans Administration Center, Los Angeles, and at St. John's Hospital, Santa Monica, Calif., from 1946 to 1952. The patients were adults whose ages ranged from 22 to 82 years at the time of operation (Table 1). Of the patients, 83 (41.5%) were women and 117 (58.5%) were men (Table 2). The high percentage of men in this series was due to the preponderantly male population of the Veterans Administration Hospital. Only 20% of the patients from the Wadsworth Hospital were women, while 86% of those operated on at St. John's Hospital were women.

Thirteen of the patients had substernal extension of their goiter.

It is difficult to be sure that the group reported represents a true cross section of a nonendemic area, since only 9 of the 200 patients were born in California. The exact number of patients who had lived in an endemic-goiter area could not be determined from the data in the records, but it was estimated from the information available that 20% had lived in such a region.

ANALYSIS OF DATA

Carcinoma occurred in 15 (7.5%) of the 200 consecutive cases in this series (Table 3). In five of these, operation was performed, with a diagnosis of nontoxic nodular goiter, and carcinoma was found on histologic examination. In 10 cases

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. (*a*) Beahrs, O. H.; Pemberton, J. deJ., and Black, B. M.: Nodular Goiter and Malignant Lesions of the Thyroid Gland, J. Clin. Endocrinol. **11**:1157, 1951. (*b*) Cole, W. H.; Slaughter, D. P., and Majarakis, J. D.: Carcinoma of the Thyroid Gland, Surg., Gynec. & Obst. **89**:349, 1949. (*c*) Crile, G., Jr., and Dempsey, W. S.: Indications for Removal of Non-Toxic Nodular Goiters, J. A. M. A. **139**:1247, 1949. (*d*) Ward, G. E.; Hendrick, J. W., and Chambers, R. G.: Carcinoma of the Thyroid Gland, Ann. Surg. **131**:473, 1950.

a preoperative diagnosis of carcinoma had been made. In 6 of these 10 cases the diagnosis had been made at an operation previously performed at another hospital. In three it was established by biopsy of cervical lymph nodes, and in one case a clinically far-advanced malignancy was confirmed by biopsy.

TABLE 1.—*Age Incidence in 200 Patients with Thyroid Disease.*

Pathology	Age Range	Average Age
Carcinoma	24-69	50
Toxic		
Diffuse	23-52	35
Nodular	18-60	44
Nontoxic		
Diffuse	28-60	44
Nodular	22-82	49
Solitary nodules	22-82	49
Multiple nodules	22-71	49

TABLE 2.—*Sex Distribution in 200 Patients with Thyroid Disease*

Pathology	Men	Women
Carcinoma	12	3
Toxic	34	19
Diffuse	20	14
Nodular	14	5
Nontoxic	67	59
Diffuse	1	1
Nodular	66	58
Chronic thyroiditis	3	2
Normal thyroid (abnormal thyroid cartilage)	1	0
Total	117	83

TABLE 3.—*Preoperative and Postoperative Diagnosis in 200 Consecutive Cases of Thyroid Disease*

Pathology	Preoperative Diagnosis	Postoperative Diagnosis
Carcinoma	10	15
Toxic	54	53
Diffuse	39	34
Nodular	15	19
Nontoxic	135	126
Diffuse	2	2
Nodular	133	124
Chronic thyroiditis	1	5
Normal thyroid	0	1
Total	200	200

Of the patients, 54 (27%) had signs and symptoms of hyperthyroidism, and of these, 15 had exophthalmos (Table 3). With one exception, these patients were found to have either diffuse or nodular goiter. One patient was found to have chronic thyroiditis. Carcinoma was not encountered in this group. This finding is consistent with the low incidence of carcinoma in toxic goiter reported elsewhere.[2]

One hundred and thirty-five patients (67.5%) were subjected to operation because of goiter, without evidence of hyperthyroidism or carcinoma. Of this

2. Pemberton, J. deJ., and Black, B. M.: Association of Carcinoma of the Thyroid and Exophthalmic Goiter, S. Clin. North America **28**:935, 1948.

group 127 (94.1%) had no symptoms referable to the thyroid other than the presence of an enlarged gland. Thirty-six patients had not noticed the goiter, which was detected on routine physical examination. All the carcinomas occurring in this series were found in patients with nontoxic nodular goiter.

CARCINOMA OF NONTOXIC NODULAR GOITER

Because it has been stressed by several investigators that the incidence of carcinoma is significantly greater in nontoxic goiters with a solitary nodule than in goiters with multiple nodules,[3] the nontoxic nodular goiters in this series were separated into these two groups for investigation of this aspect of the problem.

When an effort is made to determine the incidence of carcinoma in cases of nontoxic nodular goiter, a standard for determining nodularity must be established. It has been noted that marked discrepancies may exist between clinical impression, operative findings, and results of pathologic examination.[4] Because of the possibility of such discrepancy, we have combined the descriptions of the surgical pathologist and the surgeon as a basis for assessing nodularity. The error in this method appears reasonably small, because lobectomy or subtotal thyroidectomy was generally employed in treating nodular goiter in this series.

Eighty-five patients who presented a clinical impression of nontoxic goiter with a solitary nodule were operated upon (Table 4). In 36, or 42%, the presence of the thyroid enlargement had been discovered during physical examination and had not been known to the patient. Clinically the nodules were described as measuring from 0.5 to 5 cm. in diameter. Carcinoma was found in four of this group of 85 patients (4.7%). The nodules that were later found to be carcinomatous had been described as being 0.5 to 4 cm. in diameter. The operative records and the pathologist's description revealed that solitary nodules were present in only 46 (54%) of the patients (Table 4). Multinodular goiter was found in 33 (39%), and 1 patient was found to have chronic thyroiditis. In one instance a normal thyroid gland was pressed forward by an abnormal thyroid cartilage.

Forty-eight patients who were operated upon presented a clinical impression of multinodular nontoxic goiter (Table 5). Upon histologic examination one of these was found to have carcinoma. In 39, or 81%, the clinical impression of multiple nodules was correct. Five, or 11%, were found to have solitary nodules. Two patients were found to have chronic thyroiditis, and in one a substernal colloid goiter was discovered.

Thus, in 133 cases of clinically nodular nontoxic goiter, unsuspected carcinoma was found in 5 instances, or 3.75%. In this group of five, three patients were men and two were women. Their ages ranged from 33 to 55.

Ten other patients in the present series had carcinoma of the thyroid, making a total of 15 with thyroid carcinoma. Twelve of these were men and three were women. Their ages ranged from 24 to 69 years, with an average age of 50 years. It is of interest that the average age of the patients with nontoxic nodular goiter

3. (*a*) Cole, W. H.; Majarakis, J. D., and Slaughter, D. P.: Incidence of Carcinoma of the Thyroid in Nodular Goiter, J. Clin. Endocrinol. **9**:1007, 1949. (*b*) Cole, Slaughter, and Majarakis.[1b] (*c*) Crile and Dempsey.[1c]

4. Lange, H. J., and Maclean, K. F.: Malignant Neoplasms of the Thyroid, Surgery **26**:862, 1949.

was 49 years, with an age range of from 22 to 81 years. Metastases to cervical nodes were found to be present in 11 of the patients with carcinoma of the thyroid, and 2 of the patients in whom carcinoma had not been suspected clinically were in this group. In one of these the primary lesion measured 5 mm. in diameter.

RADIOACTIVE IODINE

Radioactive-iodine studies on the group of patients from the Veterans Administration Hospital have been available during the past two years.[5] The methods used in these diagnostic studies have been reported elsewhere.[6] To 40 of the patients in this series, tracer doses of I^{131} were administered to determine the status of thyroid function. In 57 patients, outlines of the thyroid were obtained. These studies have been of distinct assistance in diagnosis. The use of radioactive iodine has been

TABLE 4.—*Pathologic Report in 85 Cases of Clinically Nontoxic Solitary-Nodular Goiter*

Pathology	No. Cases	%
Unsuspected carcinoma	4	5
Solitary-nodular goiter	46	54
Multinodular goiter	33	39
Chronic thyroiditis	1	1
Normal thyroid	1	1
Total	85	100

TABLE 5.—*Pathologic Findings in 48 Cases of Clinically Nontoxic Multinodular Goiter*

Pathology	No. Cases	%
Unsuspected carcinoma	1	2
Solitary-nodular goiter	5	11
Multinodular goiter	39	81
Chronic thyroiditis	2	4
Colloid goiter	1	2
Total	48	100

useful in determining the presence or absence of hyperthyroidism. One patient had a superior mediastinal mass that presented diagnostic difficulties until the mass was demonstrated by means of the scintillation counter to have affinity for radioactive iodine. A large substernal goiter was found at operation. Two patients were found to have increased uptake of radioactive iodine compatible with hyperthyroidism, and the concentration of radioactive iodine was confined to a single moderately large nodule in one lobe.

With the use of the refinements in technic and instrumentation which have been developed more recently, delineation of areas of decreased uptake of radioactive iodine within the gland has become possible.[6b] In one case there was difference of opinion among clinicians as to whether the thyroid was diffusely enlarged or

5. Dr. Franz Bauer and Dr. William Goodwin, of the Radioisotope Unit, carried out these studies.

6. (*a*) Allen, H. C., Jr.; Libby, R. L., and Cassen, B.: The Scintillation Counter in Clinical Studies of Human Thyroid Physiology Using I^{131}, J. Clin. Endocrinol. **11**:492, 1951. (*b*) Bauer, F. K.; Goodwin, W. E.; Libby, R. L., and Cassen, B.: Visual Delineation of Thyroid Glands in Vivo, J. Lab. & Clin. Med. **39**:153, 1952.

whether it was nodular. The scintigram demonstrated three nodules of various sizes, and this finding was confirmed at operation. This observation suggests that such studies will be of considerable aid in diagnosis, although carcinomatous nodules cannot be differentiated from benign ones.

Five of the patients in this series who had carcinoma of the thyroid with metastases have been treated with radioactive iodine. Only one of these had sufficient concentration of I^{131} in the metastatic lesions to make therapy appear to be of possible benefit.

TREATMENT

The problems encountered in the surgical treatment in cases of nodular nontoxic goiter were our primary concern in this study. Operations for this disease should remove the goiter, alleviate symptoms related to its presence, assure as well as possible against its recurrence, and provide adequate local treatment should an unsuspected neoplasm be present. At the same time, the morbidity should be low, and sufficient glandular tissue should be left in place to prevent postoperative myxedema. The problem of how to deal with unsuspected carcinoma in nodular goiter is particularly important. It has been our experience that carcinoma is usually not recognized at the time of operation unless there is evidence of extension to surrounding structures or metastases are present. Even in some cases in which carcinoma is suspected, rapid frozen-section examination may not be conclusive. Since simple enucleation of an adenoma and partial removal of a lobe are inadquate in cases in which unsuspected carcinoma is present, it has been our policy to advocate lobectomy. Routine visualization of the recurrent laryngeal nerve prevents injury to the nerve. Lobectomy best answers the criteria previously outlined and at times provides all the operative intervention required if carcinoma is discovered in the specimen. Lobectomy includes removal of the isthmus as well.

In most cases of carcinoma of the thyroid, unilateral lobectomy will suffice if the lesion involves only a small portion of the lobe. If the tumor arises medially and involves the isthmus or if the entire lobe is involved, total thyroidectomy should be performed. Total extirpation of the thyroid is also indicated when metastases are present.

The indications for radical neck dissection are still somewhat uncertain in carcinoma of the thyroid. It is our opinion at the present time that radical neck dissection is indicated when lymph node metastasis is present and limited to the cervical region. Neck dissection should include resection of the sternocleidomastoid muscle and internal jugular vein. Seven of our patients had radical neck dissection. In five cases, palpable nodes were present. Two patients were found to have unsuspected papillary adenocarcinoma, and radical neck dissection was performed two weeks later. Cervical node metastasis was found in one of these. Cervical lymph node metastasis is frequent in papillary adenocarcinoma of the thyroid,[7] and radical neck dissection therefore appears indicated when the primary thyroid neoplasm is of this type. In other types of carcinoma, the presence of palpable nodes is an indication for neck dissection.

7. Cattell, R. B.: Indications for Neck Dissection in Carcinoma of the Thyroid, J. Clin. Endocrinol. **10**:1099, 1950. Frazell, E. L., and Foote, F. W., Jr.: The Natural History of Thyroid Cancer: Review of 301 Cases, J. Clin. Endocrinol. **9**:1023, 1949.

COMMENT

There appears to be general agreement that carcinoma seldom occurs in patients with toxic goiter, whether nodular or diffusely enlarged. This finding has been supported in the present series. Controversy centers about the problem of whether carcinoma occurs sufficiently often in nontoxic nodular thyroid enlargement to warrant operative intervention merely on the basis of finding such a gland on examination.

Most of the reports on this subject have appeared from clinics in areas in which goiter is endemic, and it is felt that more information should be obtained from parts of the country where goiter is not as prevalent.

In the present series 133 patients were clinically considered to have nontoxic nodular goiters, and 5 of these were found to have carcinoma which had not been suspected either clinically or at the time of operation. It is perhaps of greater significance that four of the carcinomas were found in patients who were unaware of thyroid disease until the presence of nodular enlargement was discovered at the time of physical examination. There was no operative mortality in this series, and

TABLE 6.—*Distribution of Thyroid Carcinoma in Nontoxic Nodular Goiter*

Pathology	No. Cases		No. Carcinoma		% Carcinoma	
	Men	Women	Men	Women	Men	Women
All nontoxic, nodular goiters	79	63	12	3	15.2	4.8
Nontoxic, solitary nodules	46	18	10	1	21.7	5.6
Nontoxic, multiple nodules	33	45	2	2	6.1	4.7

more than half the procedures were performed by resident surgeons. It would thus appear that the danger of a goiter of the type under discussion would considerably outweigh the dangers of operation.

It is to be noted also that the ages of the patients with carcinoma paralleled the ages of those with nodular goiter, the average being essentially the same (Table 1).

The observation of Ward and associates [1d] that the relative incidence of carcinoma in nontoxic nodular goiters is greater in men than in women has been borne out in the present series. Of the 15 patients with carcinoma, 12 were men, and 3 of the 5 unsuspected neoplasms were in men (Table 6). This difference in sex incidence was influenced by the predominantly male population of the Veterans Administration Hospital and perhaps does not represent a true index for the population in the area.

The present series also confirms the point emphasized by Cole and others [3a] concerning the significance of the presence of a solitary nodule in a thyroid. Although the correlation between clinical impression and actual gross pathologic findings is not accurate, the incidence of neoplasm remains high. The use of radioactive iodine in outlining the thyroid may prove to be of considerable value in this connection. In 55 glands solitary nodules were actually present, and four of these harbored carcinoma. Thus, the actual incidence of carcinoma in solitary nodules was 7.3%. In 73 glands multinodular enlargement was found, and in only 1 was it malignant (1.37%). These figures are presented to stress the dangerous potentialities of solitary nodules in goiter. It is of greater clinical significance that 5%

of the goiters thought at physical examination to have solitary nodules were later found to be the site of carcinoma. Because of these considerations, it seems reasonable to advise patients with nodular nontoxic goiter to submit to thyroidectomy.

SUMMARY

Data concerning the incidence of carcinoma in 200 consecutive patients with thyroid disease subjected to operation have been presented. The material was obtained in a nonendemic-goiter area, southern California.

In this series of 200, 15 cases of carcinoma (7.5%) were encountered and all 15 occurred in nontoxic nodular goiter.

Of 133 patients with clinically nontoxic nodular goiter, 5, or 3.75%, were found to have unsuspected carcinoma. Of the 85 patients who presented a clinical impression of solitary-nodular goiter, 4 (5%) were later found to have carcinoma, while only 1 (2%) of the 48 who were thought clinically to have multinodular goiter was subsequently found to have carcinoma. The age distribution of the patients with carcinoma did not differ significantly from that of those with nontoxic nodular goiter without carcinoma. The incidence of carcinoma was approximately four times greater in men than in women in this series.

The analysis of our data supports the findings reported from endemic-goiter regions, although the percentages are somewhat lower than those reported by Crile [1c] and Cole.[1b] The results support the opinion that operation should be advocated in cases of nontoxic multinodular goiter and should be urged in cases of solitary-nodular goiter.

PARTIAL OCCLUSION OF THE PORTAL VEIN IN EXPERIMENTAL ASCITES

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A PRELIMINARY report of our study of the reciprocal hydrostatic relationship between the portal and caval systems was published recently.[1] The present report is concerned with the specific effects of partial occlusion of the portal vein on the formation and re-formation of experimental ascites caused by partial occlusion of the vena cava in the dog.

Of the several methods employed in the past to produce ascites in the dog, partial occlusion of the inferior vena cava above the diaphragm produces ascites most closely mimicking that seen following hepatic cirrhosis in the human. Sudden complete occlusion of the hepatic veins in the dog will result in death in from 10 to 60 minutes.[2] Partial blockage of the hepatic veins is difficult to accomplish in the dog because these veins are numerous and short. However, congestion of the liver and ascites can be produced consistently in two to three weeks by partial occlusion of the vena cava just above the entrance of the hepatic veins. This technique was used by Whipple and Sperry,[3] who used sutures to constrict the vena cava above the diaphragm. Bolton [4] caused ascites in cats by constricting the pericardium around the right auricle and later [5] used metal bands about the vena cava above the liver.

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2. Child, C. G., III; McClure, R. D., Jr., and Hays, D. M.: Studies on Hepatic Circulation in Macaca Mulatta Monkey and in Man, in Surgical Forum: Clinical Congress of the American College of Surgeons, Philadelphia, W. B. Saunders Company, 1951, pp. 140-146.

3. Whipple, G. H., and Sperry, J. A.: Chloroform Poisoning: Liver Necrosis in Repair, Bull. Johns Hopkins Hosp. **20**:278-289, 1909.

4. Bolton, C. J.: Experimental Production of Uncompensated Heart Disease with Especial Reference to Pathology of Dropsy, J. Path. & Bact. **9**:67-86, 1903.

5. Bolton, C. J.: Further Observations on Pathology of Dropsy, J. Path. & Bact. **20**:290-326, 1916.

Parsons and Holman [6] showed that partial constriction of the vena cava anywhere between the right auricle and the diaphragm would produce ascites. Zimmerman and Hillsman [7] used an aluminum band for this purpose in 1930. McKee and his associates [8] have published the illustrated technique for the procedure of partial occlusion of the vena cava by means of an aluminum band and have reported rather extensive studies [9] on the experimental ascites so produced. Volwiler, Bollman, and Grindlay [10] have described another type of experimental ascites which they produced by placing cellophane bands simultaneously around the portal vein and the abdominal segment of the inferior vena cava. Ascites did not develop spontaneously in these animals, but after the plasma protein concentration was markedly lowered by plasmapheresis, ascites of low specific gravity appeared before peripheral edema developed.

We have found the technique used by McKee reliable for the production of experimental ascites and have used it in this and other experiments [11] to determine the effects of various procedures upon the formation and course of ascites in the dog.

Experimental procedures to alter the course of such an ascites must be undertaken either at the time the lesion is produced or within four months afterward, since the ascites disappears spontaneously in from four to six months. Spontaneously recovered animals regain a rather normal appearance over a period of several weeks. This is remarkable in view of the fact that while the ascites is present these animals are markedly emaciated. The formation of a large number of collateral venous channels is probably responsible for the eventual spontaneous absorption of the ascitic fluid.

It is well known that dogs, cats, and rabbits will not survive sudden acute ligation of the portal vein.[2] However, Child [12] has shown that the Macacus mulattus

6. Parsons, H. G., and Holman, E.: Experimental Ascites, in Surgical Forum: Clinical Congress of the American College of Surgeons, Philadelphia, W. B. Saunders Company, 1950, pp. 251-258.

7. Zimmerman, H. M., and Hillsman, J. A.: Chronic Passive Congestion of Liver: Experimental Study, Arch. Path. **9**:1154-1163, 1930.

8. McKee, F. W.; Schilling, J. A.; Tishkoff, G. H., and Hyatt, R. E.: Experimental Ascites: Effects of Sodium Chloride and Protein Intake on Protein Metabolism of Dogs with Constricted Inferior Vena Cava, Surg., Gynec. & Obst. **89**:529-540, 1949.

9. McKee, F. W.; Schloerb, P. R.; Schilling, J. A.; Tishkoff, G. H., and Whipple, G. H.: Protein Metabolism and Exchange as Influenced by Constriction of Vena Cava: Experimental Ascites an Internal Plasmapheresis; Sodium Chloride and Protein Intake Predominant Factors, J. Exper. Med. **87**:457-471, 1948. McKee, F. W.; Hyatt, R. E.; Wilt, W. G., Jr.; Tishkoff, G. H., and Whipple, G. H.: Protein Metabolism and Exchange as Influenced by Constriction of Vena Cava, ibid. **90**:447-459, 1949. McKee, F. W.; Wilt, W. G., Jr.; Hyatt, R. E., and Whipple, G. H.: Circulation of Ascitic Fluid: Interchange of Plasma and Ascitic Fluid Protein as Studied by Means of C^{14}-Labelled Lysine in Dogs with Constriction of the Vena Cava, ibid. **91**:115-122, 1950. McKee.[8]

10. Volwiler, W.; Bollman, J. L., and Grindlay, J. H.: Comparison of 2 Types of Ascites, Proc. Staff Meet., Mayo Clin. **25**:31-33, 1950

11. Laufman, H.; Ross, A.; Bernhard, V. M.; Bourdeau, R. V.; Douglass, T. C., and Furr, W. E., Jr.: Graded Hepatic Arterial Ligations in Experimental Ascites, to be published. Laufman, H.; Ruge, D.; Ross, A., and Bernhard, V. M.: Sympathectomy in Experimental Ascites, to be published. Laufman, H.; Bernhard, V. M., and Ross, A.: Massive Intestinal Resection in Experimental Ascites, to be published. Laufman.[1]

12. Child, C. G., III; Milnes, R. F.; Holswade, G. R., and Gore, A. L.: Sudden and Complete Occlusion of Portal Vein in Macaca Mulatta Monkey, Ann. Surg. **132**:475-495, 1950.

monkey will usually survive uneventfully the complete and sudden occlusion of the portal vein. Child [13] has also done portal occlusion and resection in man in the course of radical pancreatectomy. In none of Child's monkeys could ascites or enlargement of the spleen be detected clinically or at reoperation. Complete occlusion of the portal vein in the dog can be accomplished only in stages if one is to expect survival. Douglass and co-workers,[14] Morris and Miller,[15] and Peck and Grover [16] have produced gradual occlusion of the portal vein over a two- to three-week period without producing a consistent elevation of portal venous pressure or ascites. It appears, therefore, that portal hypertension with ascites can only be produced by intrahepatic or suprahepatic venous blockage and not by infrahepatic occlusion.

Davis and Tanturi [17] partially occluded the portal vein with a Goldblatt clamp as a means of reducing the systemic blood pressure of dogs previously rendered hypertensive by renal constriction. They found that the occlusion had to be sufficient to result in fatty degeneration of the liver in order to reduce the hypertension. Peck and Grover [16] are of the opinion that at least part of this effect is due to some other factor influenced by the anterior pituitary and originating within the splanchnic mesentery. Grindlay and Bollman [18] studied regeneration of liver in the dog after a partial hepatectomy and showed that severe constriction of the portal vein reduced the ability of the liver to regenerate after resection. However, if the vena cava was constricted below the liver simultaneously with the portal vein constriction, good regeneration was observed. They postulated that the liver needs to be well filled with venous blood for good regeneration to occur.

These workers placed cellophane® bands on the vena cava above the diaphragm and on the portal vein at the hilum of the liver.[19] With the unpredictable rate of constriction so produced, they obtained ascites in all their animals. We repeated this work as part of our preliminary program and found that ascites formed much more slowly when cellophane® bands were placed on both the thoracic vena acva and portal vein than it did in animals with a cellophane® band on the vena cava alone.

THEORETICAL CONSIDERATIONS

We reasoned that if partial occlusion of the vena cava above the diaphragm results in sufficient congestion of the liver to produce ascites, it might actually be

13. Child, C. G., III; Holswade, G. R.; McClure, R. D., Jr.; Gore, A. L., and O'Neill, E. A.: Pancreaticoduodenectomy with Resection of Portal Vein in Macaca Mulatta Monkey and in Man, Surg., Gynec. & Obst. **94**:31-45, 1952.

14. Douglass, T. C.; Mehn, W. H.; Lounsbury, B. F.; Swigert, L. L., and Tanturi, C. A.: Attempts at Experimental Production of Portal Hypertension, A. M. A. Arch. Surg. **62**:785-788, 1951.

15. Morris, A. N., and Miller, H. H.: Chronic Portal Vein Occlusion and Portal Hypertension in Dog, Surgery **30**:768-774, 1951.

16. Peck, M. E., and Grover, R. F.: Cardiovascular Responses to Acute Ligation of Portal Vein, A. M. A. Arch. Surg. **64**:665-678, 1952.

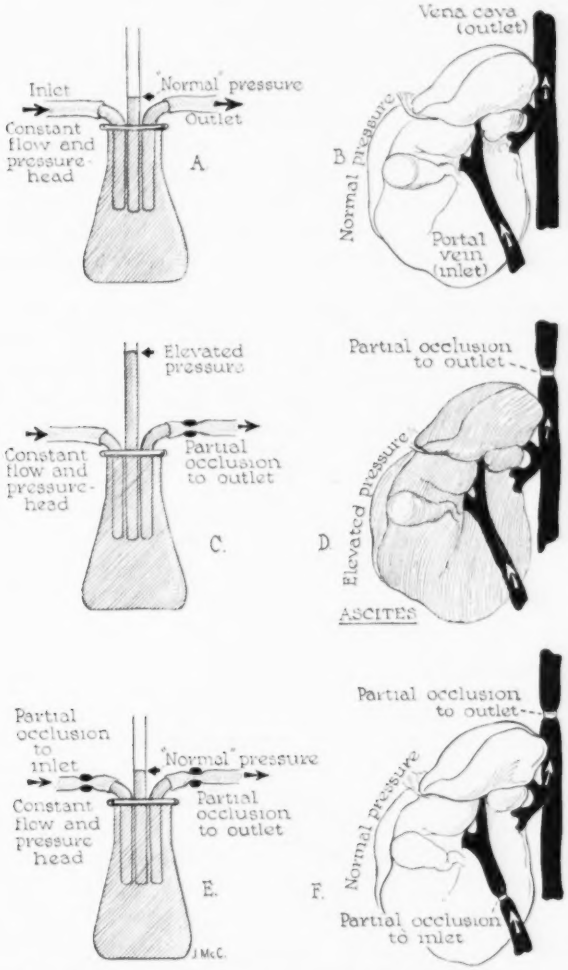
17. Davis, L., and Tanturi, C. A.: Liver as Factor in Experimental Renal Hypertension, A. M. A. Arch. Surg. **62**:325-334, 1951.

18. Grindlay, J. H., and Bollman, J. L.: Regeneration of Liver in Dog After Partial Hepatectomy: Role of Venous Circulation, Surg., Gynec. & Obst. **94**:491-496, 1952.

19. Gray, H. K.: Clinical and Experimental Investigation of Circulation of the Liver, Ann. Roy. Coll. Surgeons England **8**:354-365, 1951.

possible to prevent this congestion by diminishing the venous flow into the liver by measured constriction of the portal vein.

To test our hypothesis we built a working model, the essential features of which are shown in the Figure. The model consisted of a flask representing the liver, with a source of constantly flowing colored fluid under a constant pressure head



Working model compared with alterations in venous inflow and outflow of liver. *A* and *B*, normal situation. Manometer in flask registers "normal" intrahepatic pressure when inflow and outflow are unaltered. *B* and *C*, when partial occlusion to outflow is made, pressure within liver rises as does fluid level in manometer tube. *D* and *E*, when both inflow and outflow are equally occluded, intrahepatic pressure approaches normalcy, as shown by adjustment of fluid level in manometer tube.

entering the flask from one side and leaving it on the other by way of compressible plastic tubing. A vertical glass tube in the flask served as a manometer to measure pressure within the flask (liver). When a partially occluding clamp was placed

on the outflow tube (hepatic vein or vena cava), the pressure within the flask rose since the rate of flow and initial pressure head were constant. If, then, another partially occluding clamp was placed on the inflow tube (portal vein), the pressure within the flask readjusted itself to "normal" levels. A motion picture was made showing this apparatus in operation and demonstrating the mechanisms described above.[1]

ANIMAL EXPERIMENTS

Our next step was to apply the principle of the working model to the living animal. With the use of positive-pressure anesthesia and the usual aseptic surgical precautions and with the animals given intravenous injections of pentobarbital (nembutal*) anesthesia, an aluminum band was placed about the vena cava above the diaphragm, occluding the vein to about 50% of its diameter. In the 10 dogs in which this was the only procedure, the result was an almost immediate increase in the caval pressure below the band from a control level of about 4 cm. water to 12 to 20 cm. of water. These dogs were used as controls. Chemical studies (Table 1)

TABLE 1.—*Partial Constriction Inferior Vena Cava in Thorax (Control, Dog 139)*

Date	Operation	Total Protein	Alb.	Glob.	Sodium	Ascites*	Weight, Lb.
6/27/51	Vena cava constriction	7.0	2.7	4.6	186	.....	25
7/13/51	Abdominal exploratory	5.15	2.65	3.5	148	3,000	30
7/25/51	.....	5.3	2.75	2.55	135	+	..
8/ 9/51	.....	5.7	2.7	3.0	145	++++	..
8/22/51	.....	5.1	2.75	2.35	147	++++	27.5
9/ 4/51	.....	5.8	3.3	2.5	152.5	++++	..
10/ 9/51	.....	6.6	2.95	3.75	143.5	++++	..
11/ 6/51	.....	6.2	2.55	3.65	161	++	..
12/11/51	.....	6.4	3.35	3.05	163	.....	..
12/19/51	.....	5.6	3.1	2.5	164	+	22
1/22/52	.....	5.9	3.4	2.5	157.5	.....	..
3/18/52	.....	5.9	2.95	2.95	151	.....	..
5/ 5/52	.....	7.0	2.9	4.1	156.5	.....	22

* Based on correlation studies: + = < 500 cc., ++ = 1,000 to 1,500 cc., +++ = 2,000 to 2,500cc., ++++ = > 3,000 cc. of fluid.

coincided very closely with the work of McKee and associates.[8] Within two to three weeks a rather large ascites occurred ranging in volume from 1,800 to 3,000 cc., and the animals became progressively emaciated over the next several months. In general, the procedure resulted in a diminution in the plasma protein levels and an increase in sodium levels. In four to six months the ascites gradually diminished in the surviving control animals. It was interesting to note that the serum protein levels adjusted themselves to normalcy without the use of high protein diets. The serum sodium levels, however, remained elevated long after the ascites had disappeared.

In a group of 20 animals the caval band was applied as usual, after which the thoracic incision was closed and an abdominal incision made immediately to expose the portal vein. Portal vein pressures after the band had been placed on the vena cava in the chest were in the vicinity of 16 to 30 cm., instead of the expected normal levels of 6 to 11 cm. A Goldblatt clamp was then placed about the portal vein constricting it about 50%, never allowing it to become tight enough to cause color change in the bowel. After the application of the Goldblatt clamp, pressure within

the portal vein above the clamp was reduced to 6 to 13 cm. of water. In no instance did the application of the clamp cause any change in arterial (femoral) pressure. The result in these animals was the prevention, or almost complete prevention, of ascitic-fluid formation for from 3 to 12 weeks, provided the clamp was placed above the gastroduodenal vein. If the clamp was placed below the gastroduodenal vein, ascitic fluid would re-form within two weeks. We observed the latter situation in our first four animals, which are not included in our results.

Five of the 20 animals in this group died between the fourth and sixth postoperative week from intercurrent infection. None of these animals had ascites at the time of death. Surviving animals usually showed no ill effects other than an altered course of ascites formation.

In six instances, some ascites (up to 500 cc.) developed after three weeks, was short-lived, and was resorbed spontaneously within two weeks. Small amounts

TABLE 2.—*Simultaneous Application of Thoracic Caval Band and Goldblatt Clamp on Portal Vein (Dog 152)*

Date of Surgery	Total Protein	Alb.	Glob.	Sodium	Ascites*	Weight, Lb.
8/ 1/51	6.7	4.2	2.5	146	0	15
8/15/51	6.55	3.45	3.1	158	0	15
8/28/51	6.2	3.4	2.8	149	++	15
9/18/51	6.1	2.5	3.6	155	++	..
9/26/51	7.4	3.6	3.8	140	0	..
10/ 9/51	6.5	3.65	2.85	146	0	..
10/23/51	5.9	3.5	2.4	162	0	..
11/ 6/51	5.9	3.25	2.65	163	+	..
11/20/51	6.0	3.1	2.9	169	++	..
12/ 4/51	5.2	2.75	2.45	172	++	..
12/21/51	5.45	3.3	2.15	163	+	..
1/ 4/52	6.55	3.4	3.15	169.5	0	..
1/15/52	6.5	3.5	3.0	176	0	..

* Based on correlation studies: + = < 500 cc., ++ = 1,000 to 1,500 cc. of fluid.

formed again after 12 to 14 weeks and again disappeared (Table 2). In five others, ascites re-formed after six weeks to almost the degree seen in control animals (2,000 to 3,000 cc.). These animals were killed for postmortem studies, which revealed a nutmeg-type liver, the result of a chronic passive congestion. In the remaining four animals no discernible ascites developed at any time, and, except for a moderate degree of emaciation, they appeared perfectly well. We ascribed the loss of weight to poor absorption from the gastrointestinal tract due to the constriction of the portal vein. Thus, not counting the ascites-free animals, which died in four to six weeks, 10 of 20 surviving animals with simultaneous partial occlusions above and below the liver had no ascites or temporary accumulations of small amounts during the follow-up period of four months. This was striking when compared with the massive ascites lasting from four to six months in the control group.

Our next experiment dealt with the question of whether an already established ascites could be treated by partial occlusion of the portal vein. We therefore set up three groups of nine animals each in which ascites was produced by partial constriction of the vena cava and subjected them to partial occlusion of the portal vein one, two, and three weeks after the initial lesion was produced. In these animals the operation consisted of evacuating the already formed ascitic fluid, measuring it,

and applying the Goldblatt clamp to the portal vein, as described above. In each of these animals the lymphatics surrounding the portal vein were found to be greatly engorged, and evidence of early scar formation about the dilated portal vein was seen.

The results in these animals were not definitive. If only a small amount of ascites formed prior to the secondary operation, the re-formation of ascites was usually prevented. However, if a large amount of ascites had been present, the operation of partial constriction of the portal vein was usually not successful. The best results were obtained in the one- and two-week groups. Failure occurred in animals reoperated upon three weeks after the initial procedure, but the course of ascites was usually altered; the re-formation of ascites was usually delayed somewhat, reaching a clinically demonstrable stage in four to six weeks, rather than in two weeks. After six weeks, these animals had large accumulations of fluid appearing no different from the control group with a large ascites. Portal pressures below the Goldblatt clamp in these animals were not appreciably elevated despite the presence of ascites.

From the results in these animals we were led to the assumption that ascites, once formed, is perpetuated by factors other than congestion alone. It is logical that the profound changes in salt metabolism and other factors, perhaps endocrine, cardiac, and renal, should continue to play their roles in the perpetuation of the ascitic state once it exists, even though a mechanical factor is partially corrected.

COMMENT

The formation of ascites in our experimental animals resulted from the production of a state of portal hypertension in conjunction with congestion of the liver. Once ascites was established, it was no longer necessarily commensurate with the degree of increased pressure. Thus, our attempts at curing ascites by portal vein constriction were less and less successful the longer the ascites was present, despite the fact that the portal constriction reduced the portal pressure within the liver.

One must not overlook the changes in cardiac dynamics which undoubtedly occur with a preparation such as we used. In our earlier work a few animals died within 24 hours following application of the aluminum band too tightly about the vena cava. Postmortem studies in these animals were noncontributory. We surmised that the situation produced acute heart failure by suddenly diminishing the cardiac output, in response to a drastic reduction in venous return to the heart. In chronic heart failure the decompensatory phenomena parallel very closely those of our animals, with regard to salt retention and fluid accumulation, although the nature of the ascitic fluid is different. A further factor in salt retention may be the engorgement or passive congestion of the kidneys produced by obstruction to their venous drainage.

From our postmortem studies on dogs we know that a partial occlusion may frequently lead to a complete occlusion by thrombus or fibrosis. These animals were usually emaciated. Perhaps the cause of the emaciation was related to the portal occlusion which led to hepatic insufficiency and lack of absorption of intestinal foodstuffs by the liver.[20] Complete, sudden ligation of the portal vein in dogs is fatal,

20. Longmire, W. P., Jr.: Esophageal Bleeding, Editorial, Surg., Gynec. & Obst. **94**:757-760, 1952.

yet in monkeys and, perhaps, in man it may serve as an actual therapeutic measure in ascites. We have planned such experiments on monkeys.

Complete ligation of the vena cava at the site of a previous partial occlusion above the diaphragm can be done after two to three months following partial occlusion without causing mortality and without appreciably altering the course of the ascites or its absorption.[11] This is undoubtedly due to the establishment of collateral venous channels, which substitute for the vena cava. Two of our dogs died of pulmonary embolism six weeks after application of the thoracic caval band. The origin of the thrombus was at the site of occlusion. Occasionally, ascites did not develop in the dog after the application of the band on the thoracic vena cava. In such instances, one can only assume that there were collateral venous channels which substituted for the vena cava and by-passed the occlusion. This situation might minimize the striking results in a few of our ascites-free animals in which the bands were placed simultaneously on the vena cava and on the portal vein, since in those animals ascites may not have developed in the first place. However, in our control animals with thoracic caval bands, in only 2 of 12, ascites did not develop (only the 10 animals with ascites were studied in the control group), while in animals with the simultaneous bands above and below the liver the course of the accumulation of ascites was consistently altered.

We wish to mention another study on these animals, which will be reported fully elsewhere, in which the administration of a high salt diet to otherwise nonascitic dogs with bands above and below the liver resulted in the formation of ascites. When the high salt diet was withdrawn, the ascites disappeared. McKee's work with low salt diets in the presence of a band-induced ascites indicates that there is a profound influence on the state of ascites by salt intake.[8] Low salt intake reduced the degree of ascites in the presence of an obvious portal hypertension in his animals. An interesting observation of ours was that hypoproteinemic levels adjusted themselves spontaneously without the aid of high protein diets as ascites diminished or disappeared.

We believe our experiments demonstrate that within certain limits there is a reciprocal pressure relationship between the portal and caval systems. Once ascites is established, however, partial reduction of the portal inflow to the liver is not sufficient to reverse the process.

HYDRONEPHROSIS: EVALUATION OF PYELOPLASTY IN THE TREATMENT OF URETEROPELVIC OBSTRUCTIONS

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THE LAST decade has seen an ever-increasing interest among urologists in the subject of pyeloplasty. More widespread knowledge and experience with the many procedures in vogue, together with the aid of antibiotic and chemotherapeutic agents, have unquestionably improved the outlook in hydronephrosis. Pyeloplasty is being utilized with greater confidence and success, and because of this fact, fewer hydronephrotic kidneys are being subjected to nephrectomy.

However, there is still need for further evaluation of end-results and of the many ingenious techniques of pyeloplasty. There is still wide divergence of opinion as to the choice of treatment in ureteropelvic obstructions. The final answer has not been given as to which is the best type of pyeloplasty for a given type of obstruction.

During the past five years, 54 patients with hydronephrosis due to ureteropelvic obstructions have been operated on at Letterman Army Hospital. In 30, or 56%, of these patients, the condition had progressed to such a point that nephrectomy was considered the procedure of choice, whereas 25 conservative operations were done on the remaining 24 patients. This study concerns the 24 patients upon whom pyeloplastic procedures were done.

INCIDENCE

In this series there were 18 males and 6 females. It must be noted that in this hospital the patient load is preponderantly male. In 12 patients the condition occurred on the right, in 11 on the left, and in one the involvement was bilateral. In 4 cases there was coexistent nephrolithiasis. The average age of the patients was 26.5 years, the youngest being aged 12 years and the oldest 71. Twenty-two of the 24 patients were between 19 and 45 years of age, 15 in the third decade. This parallels the age incidence found by one of us (T. E. G.) in civil practice.

Seven patients demonstrated 10 or more pus cells per low power field from the involved side. The blood chemistry was normal in all but one patient, a 25-year-old woman with a markedly infected hydronephrotic solitary kidney and a nonprotein nitrogen level of 44 mg. per 100 cc.

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ETIOLOGIC FACTORS

All types of extrinsic and intrinsic factors were present. Usually there was more than one obstructive factor. Obstruction with high insertion occurred in 5 cases and with low insertion in 20. This is not in agreement with Culp's statement [1] that "two-thirds of our cases of stenosis and achalasia have been associated with high insertion of the ureter." In general, when aberrant vessels existed, these alone did not account for the obstruction. Aberrant vessels were found in eight cases, in only three of which were they considered to be the primary obstructive factor. The usual cause of extrinsic obstruction is fibrous bands at the ureteropelvic junction, and these were found as the primary etiologic factor in nine cases. In another 11 cases these bands were found in association with some intrinsic factor, namely, congenital stenosis, valves, or high insertion. In only two cases was congenital stenosis of the pelvic outlet found as the sole cause.

SYMPTOMS

The commonest symptom elicited in this group was pain. This usually is localized to the involved flank, is nonradiating, and varies from mild to severe and from constant to intermittent. Of the 24 patients, 21 presented this symptom in varying degrees of severity and duration. Gastrointestinal symptoms, consisting of nausea, vomiting, and flatulence unrelated to diet, are common and usually occur with attacks of pain but may be the only presenting symptom. Three patients gave histories of total gross hematuria associated with attacks of pain: One case was discovered in the urological evaluation of a patient with cystitis, who was otherwise without complaint; one case was discovered in the "good" kidney of a patient admitted for nephrectomy of a severely traumatized opposite kidney; one case was discovered in evaluating a patient with pyuria but who was entirely asymptomatic. Urinary bladder symptoms were uncommon.

DIAGNOSIS

Diagnosis is made by pyelography. Intravenous urograms are often deceiving, and, as a general rule, confirmatory retrograde films are necessary. When retrograde studies are made, these should be delayed until the day before surgery and the catheter left indwelling to reduce the possibility of infecting a poorly draining hydronephrotic kidney. A delayed film is important to demonstrate retention. Function tests are unreliable. It is usually impossible to ascertain the etiological factor except at surgery. Evidence of a thick renal parenchyma on urograms favors conservative surgery. We have had no experience with aortography as a method of evaluating potential salvability by demonstration of the renal blood supply, but it definitely offers possibilities.

TREATMENT

From the purely technical standpoint the surgeon strives for two objectives: (1) removal of the obstructive factors in the simplest manner consistent with a successful outcome, and (2) splinting or intubation of the ureteropelvic junction long enough to insure the permanent patency of the reconstructed area and prevent recurring distortion and stricture formation.

1. Culp, O. S.: Pelvic Flap Operation for Certain Types of Ureteropelvic Obstruction: Preliminary Report, Proc. Staff Meet., Mayo Clin. **26**:483-488, 1951.

The well-trained urologist should be familiar with the many surgical procedures applicable to the ureteropelvic junction, since it is only at the operating table that he will discover the causative obstructive factor and must be prepared to perform the simplest and most effective operation. In case of an obviously destroyed kidney, nephrectomy is certainly the procedure of choice (Fig. 1). Hinman [2] deplores conservative surgery when the involved kidney can never alone be capable of performing total function in the event of loss of its mate. Only when the hydronephrotic kidney is capable of performing one-fourth to one-fifth of the total function, with the potentiality of hypertrophy in the event of disaster to the other kidney, is it worth saving. Compensatory hypertrophy in the opposite kidney precludes good function in the involved organ. However, if there is bilateral impairment of function, an attempt should be made to salvage a kidney even though it has minimal function.

The various operations advocated, numbering approximately 25, have been adequately described elsewhere and will not be discussed in this paper. The principal

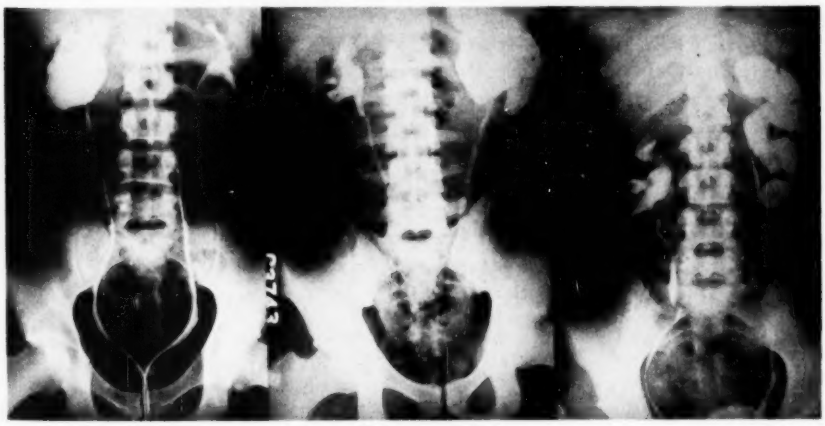

Fig. 1.—Roentgenograms taken in cases of hydronephrosis due to ureteropelvic obstruction and not amenable to conservative surgery. Marked renal destruction with compensatory hypertrophy of the opposite mate precluded pyeloplasty. Nephrectomy was performed in each case.

types of pyeloplasty in general use were reviewed by Gibson in 1945.[3] The most notable recent addition is the ingenious pelvic flap operation described by Culp, in 1951.[1] He reported on 12 patients successfully operated upon by this method.

More and more we have come to favor the Davis-Rammstedt intubation operation, when applicable, because of its simplicity and the excellent functional results obtained.[4] Davis' technique is followed, except that we prefer to bring the splinting

2. Hinman, F.: Treatment of Hydronephrosis, Southwestern Med. **26**:357-368, 1942; Surgical Treatment of Hydronephrosis, Surgery **20**:337-359, 1946.

3. Gibson, T. E.: Hydronephrosis: Classification and Plastic Repair of Ureteropelvic Obstructions, Surg., Gynec. & Obst. **80**:485-496, 1945.

4. Davis, D. M.: Intubated Ureterotomy: A New Operation for Ureteral and Ureteropelvic Stricture, Surg., Gynec. & Obst. **76**:513-523, 1943. Davis, D. M.; Strong, G. H., and Drake, W. M., Jr.: Intubated Ureterotomy: Experimental Work and Clinical Results, J. Urol. **59**:851-862, 1948. Davis, D. M.: Intubated Ureterotomy, South. M. J. **44**:275-285, 1951.

and drainage tubes out through a nephrostomy. We are not in agreement with Deming's argument that this induces infection and renal scarring, with hypertension as a later possibility.[5] In none of our cases has hypertension resulted, and the nephrostomy wound has always healed promptly after withdrawal of the tubes. In this series of patients the intubation operation was performed on seven, the Foley-Schwyzer Y-plasty being used on one,[6] the von Lichtenberg operation on two, while Deming's method of T-tube splinting was used successfully on three patients, and simple pelviureterolysis without splinting was used on two.

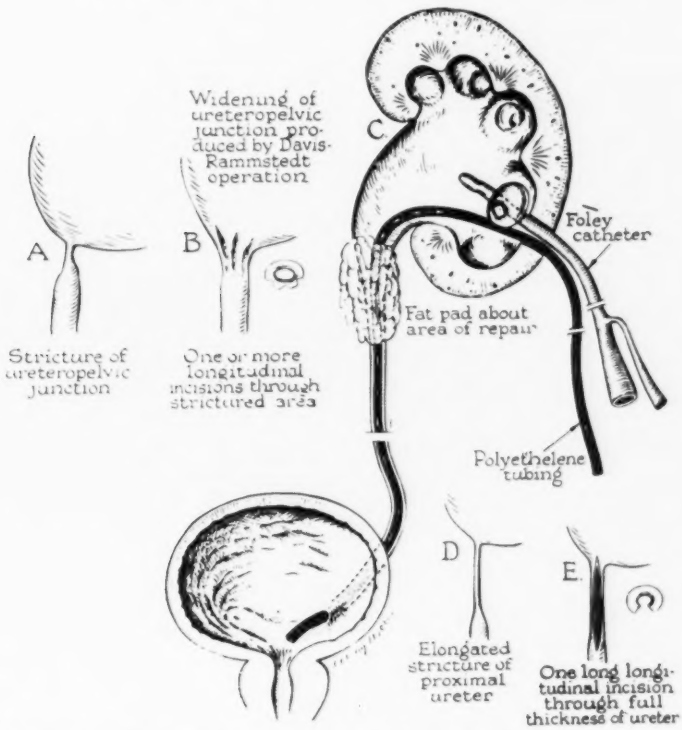

Fig. 2.—Our operation of preference for most types of ureteropelvic obstruction.

It has become axiomatic that splinting or intubation should be a necessary corollary of all pyeloplasties, unless the obstruction is of minor degree and extrinsic in type. We have come to favor the use, in general, of polyethylene tubing, utilizing the method of Davis, in which the tube runs the entire length of the ureter and out the urethra. The upper end emerges through the lower pole of the kidney alongside a Foley catheter, which drains the kidney pelvis (Fig. 2). When a simple stenosis of the pelvic outlet exists, multiple short linear incisions through the stenotic area

5. Deming, C. L.: Ureteropelvic Obstruction Due to Extrinsic and Intrinsic Lesions of the Ureter as a Clinical Entity and Its Treatment, J. Urol. **50**:420-431, 1943.

6. Foley, F. E. B.: New Plastic Operation for Stricture at the Uretero-Pelvic Junction: Report of 20 Operations, J. Urol. **38**:643-672, 1937.

down to the mucosa are sufficient. When the stenosis is more extensive, a single vertical incision is made extending through all coats of the ureter for the length of the involved portion. The diameter of the splinting tube should conform closely to the size of the ureter without causing pressure on its walls. The purpose of the splint is to act as a scaffolding and not as a dilating medium. A tight splint is likely to cause eventual fibrosis and constriction. The splint is pulled down the ureter by being fastened to a ureteral catheter, which has been passed up the ureter cystoscopically prior to operation.

In some cases the tubing causes urethral irritation when it is left in place for several weeks, and for this reason, we now make it a practice to cut off the tube as it emerges from the urethra and draw the end back into the bladder. The splint is left indwelling for two to eight weeks, depending upon the severity of the case. Several small holes cut in the splinting tube where it lies in the pelvis favor drainage. A small leather punch has been found useful in making small round holes in the tubing at the proper level. This is important in polyethylene tubing which is thin walled and likely to collapse or buckle if large oval apertures are made. Polyvinyl tubing is better in this respect, since it is thicker walled, but has the disadvantage of having a smaller lumen.

We have not used the Cumming or McIver type of combination splint and nephrostomy tube because of an early experience in which the splinting effect was lost by the tube buckling and crawling back into the pelvis. This could be obviated by excising redundant pelvis. However, we feel securer when two separate tubes are used, one for splinting and one for nephrostomy drainage, which insure better drainage and permit separate removal. In our early experience we used in some cases a pyelostomy T-tube splint, which was not too satisfactory because of its buckling propensities. The elongated T-tube of Aberhart [7] has certain desirable features, but we have had no experience with it. The use of the ureterotomy T-tube splint is favored by Deming but frowned upon by Davis.

Incrustations in the tubing have not been troublesome since the plastic tubings have come into use. Prior to the removal of the splint, dye is injected and x-rays taken to reveal the outline of the ureter. The splint is removed before the nephrostomy drain, the latter being clamped for 24 hours to ascertain patency of the new ureteropelvic junction before its removal.

Even though a definite extrinsic type of obstruction is discovered at surgery, the pelvis is usually opened and the internal orifice calibrated. Combined types of obstruction are not infrequently found. When a pelvis is definitely redundant, a partial excision is performed. Sutures are used sparingly, it being our conviction that they favor infection and necrosis. When aberrant vessels exist, they should be clamped and the kidney observed for infarction before the vessels are divided. If more than one-fifth of the kidney is infarcted, the vessels should not be divided.

When the kidney is ptosed or the ureter redundant, a Deming type nephropexy is performed after conclusion of the pyeloplasty. All wounds are drained for 48 hours with a Penrose drain. The splinting and nephrostomy tubes are brought out through the center of the flank incision to avert kinking when the patient is supine.

7. Aberhart, C.: Modified T-Tube for Use in Surgical Repair of Ureteral Lesions, J. Urol. **60**:790-791, 1948.

Fig. 3.—A white man, aged 26 years, was admitted with acute pain in right flank of six hours' duration. Similar attack lasting five days occurred one year previously. Ureteropelvic obstruction was bilateral. Many white blood cells and Pseudomonas aeruginosa were present from the right side; the left side showing no abnormalities. Left pyeloplasty was performed on June 16, 1948. Two large aberrant vessels were divided. The internal orifice, calibrated through a pyelotomy, was normal. No splinting was used, and a Deming nephropexy was performed. The right side was operated on three weeks later and a larger hydronephrosis found with similar aberrant vessels. The internal orifice was pinpoint in size. The vessels were divided, and Davis intubation was used for four weeks. Upon discharge, the patient was asymptomatic, and the urine was sterile. The above roentgenograms demonstrate the value of splinting.

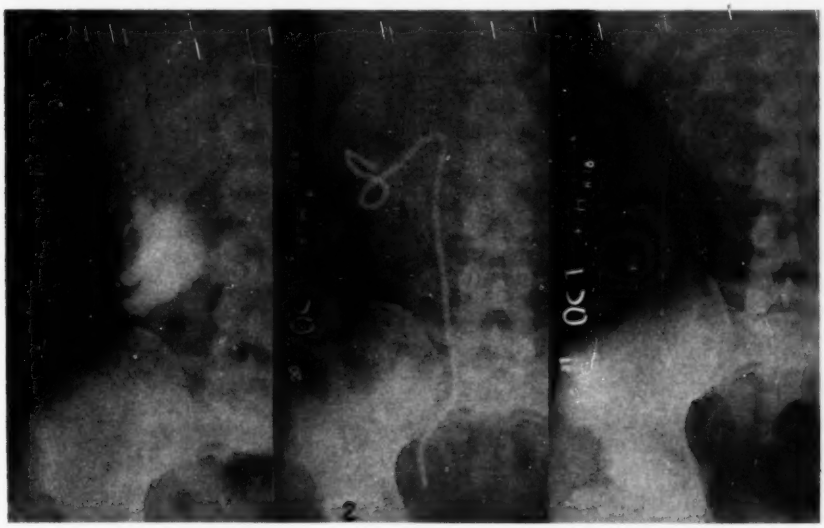

Fig. 4.—A white man, aged 23, gave a history of recurrent attacks of pain in right flank since childhood. On Oct. 8, 1947, a right pyeloplasty was performed. Dense extrinsic fibrous adhesions about the ureteropelvic region were freed. There were no aberrant vessels and no intrinsic obstruction. Through a pyelotomy a 14 F. Levin tube was inserted for ureteral splinting and left in place for three weeks. The patient made an excellent recovery, with normal urine and post-operative pyelogram.

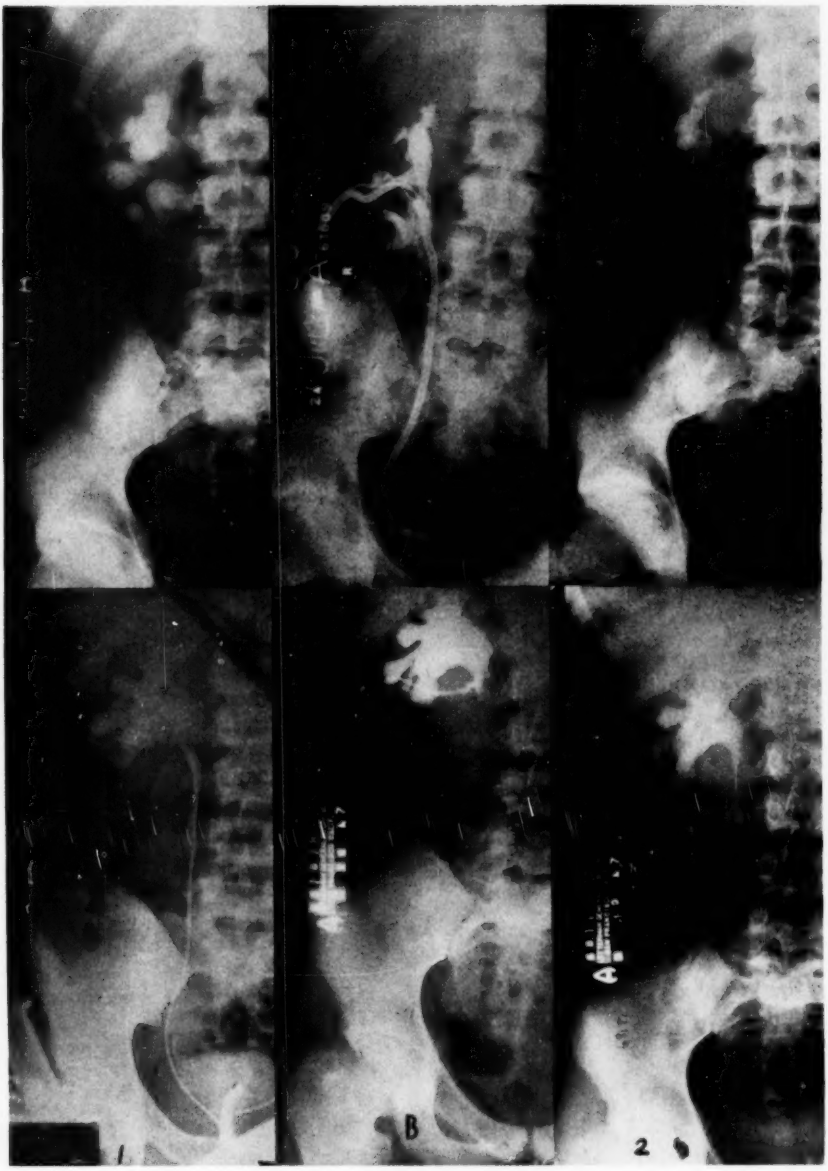

Fig. 5.—*A*, a white man, aged 26, had symphysiotomy and left pyeloplasty for horseshoe kidney in February, 1947, which was followed by left nephrectomy in April, 1947, due to severe complicating pyonephrosis. Right nephropexy was performed elsewhere February, 1948, with disastrous results. A massive retroperitoneal abscess developed, with persistent urinary flank fistula. On June 30, 1948, the right flank was explored. The abscess was drained, the fistulous opening in the pelvis closed, and a thickened ureter freed with difficulty from dense adhesions. Davis intubation was employed for seven weeks. When discharged on Nov. 12, 1948, the patient, who was almost moribund on admission, was asymptomatic. The flank was well healed. The pyelogram showed no abnormalities and the urine was sterile.

B, a white youth, aged 19 years, was admitted Jan. 8, 1947, with a history of left flank trauma and gross hematuria two months previously. Urograms revealed a nonfunctioning left kidney and a congenital right hydronephrosis. He admitted having attacks of pain in the right flank since childhood. On Feb. 11, 1947, a right pyeloplasty was performed through a Hess incision. A high take-off was discovered, with extrinsic fibrous adhesions about the ureteropelvic junction. The adhesions were freed and a Foley Y-V plasty done about a splinting T-tube, which was left in place 21 days. The postoperative result was considered good. On May 19, a left nephrectomy was done for an atrophic infarcted kidney secondary to the trauma.

RESULTS

A good result from surgery presupposes (1) uninfected urine; (2) freedom from symptoms, and (3) favorable pyelographic findings. Failures result from (1) removing the splint too soon; (2) leakage; (3) improper selection of a surgical procedure; (4) new stricture formation, and (5) infection. Relief from pain in itself does not denote cure, as has been pointed out by Henline and Menning,[8] since pelvic denervation alone will accomplish this. Experience has shown that long follow-up periods are not required. If the immediate follow-up at two or three months shows a good result, it is usually permanent.

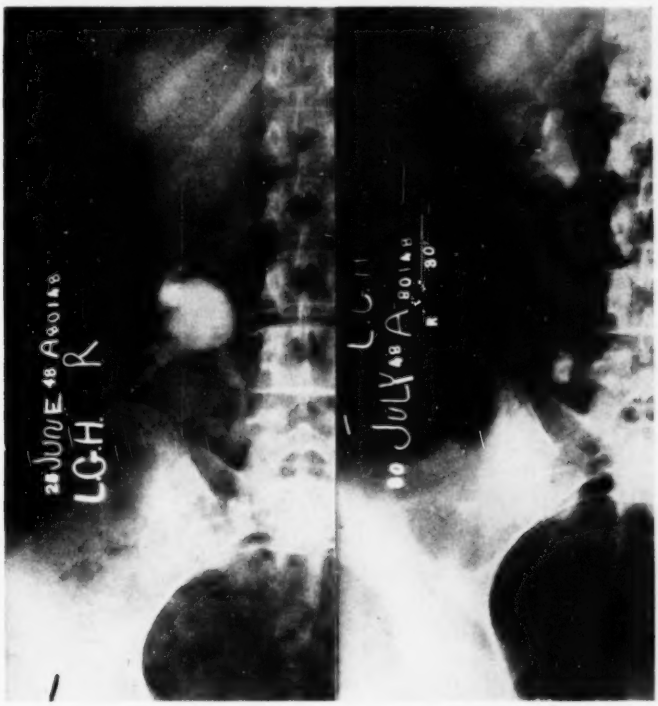

Fig. 6.—A white woman, aged 38, gave a long history of right flank discomfort. Urograms revealed a right hydronephrosis with ptosis. Exploration revealed obstructing extrinsic fibrous bands, which were divided. No intrinsic lesion was discovered. A Kidd nephropexy was done. No splinting was used. The result was considered good.

There were no deaths in this series. Good results, implying symptomatic cure, favorable functional and pyelographic findings, and freedom from infection were obtained in 72% of cases (18 operations on 17 patients). Figures 3 to 10, illustrate several of these cases. Two patients were subjected to secondary nephrectomy, one because of persistent infection and cortical abscess formation one month postoperatively, and the other because of development of a functionless pyelonephritic kidney

8. Henline, R. B., and Menning, J. H.: Management of Hydronephrosis Due to Ureteropelvic Obstruction: Preliminary Report, J. Urol. **50**:1-24, 1943.

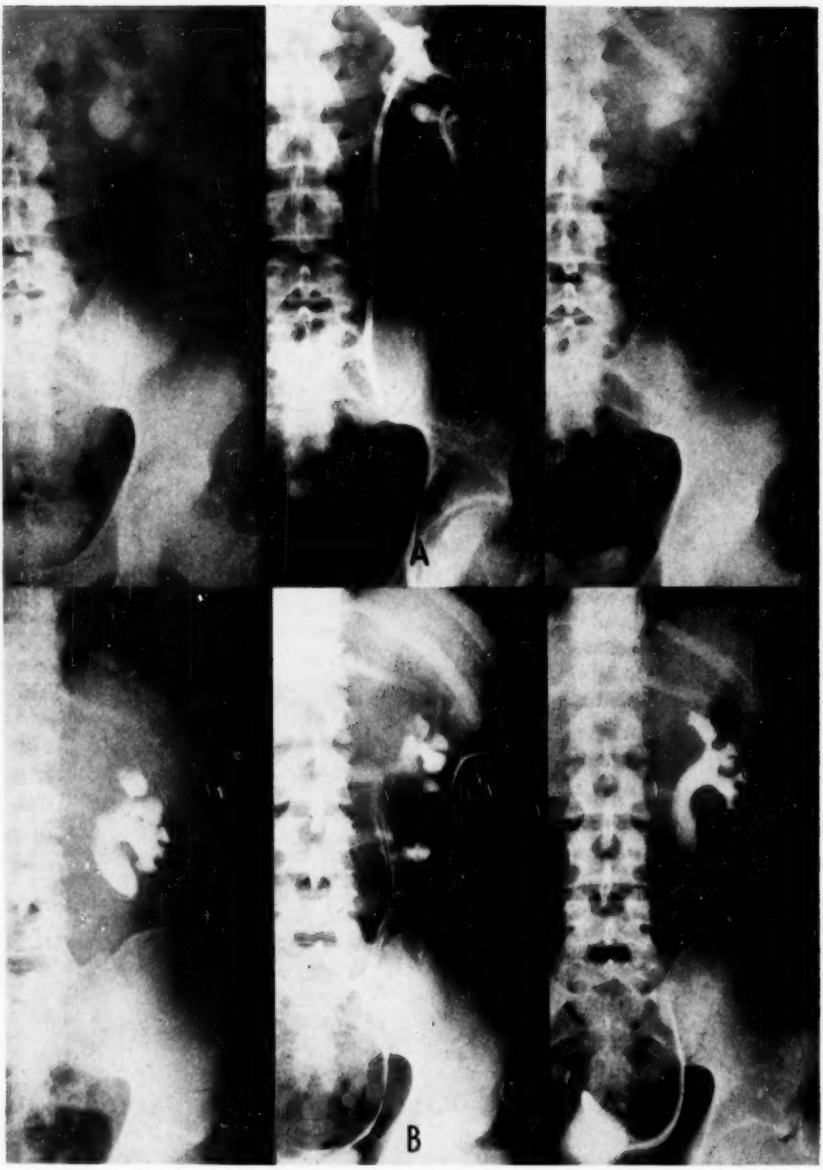

Fig. 7.—*A*, a white man, aged 27, gave a history of intermittent episodes of left flank pain, occurring particularly after ingestion of alcoholic beverages. On Oct. 9, 1950, left renal exploration revealed a moderately distended renal pelvis with multiple fibrous adhesions surrounding the ureteropelvic junction. There was no intrinsic lesion. Treatment was by pelviureterolysis, nephrostomy, and ureteral splinting for three weeks. Six months postoperatively, the pyelogram showed normal function, and the patient was asymptomatic.

B, a white man, aged 22, had one attack of radiating pain in the left upper quadrant of 24 hours' duration and associated with total gross hematuria. Left renal exploration on Dec. 18, 1950, revealed a greatly dilated pelvis, multiple fibrous adhesions at the ureteropelvic junction, a small aberrant vein, and a marked stenosis of the proximal 1 cm. of ureter. The adhesions were freed, the aberrant vessel divided, and the stenosed area incised vertically. A No. 14 F. T-tube was inserted through a distal ureterotomy and left in place 24 days. Nephrostomy drainage was not used. The patient was asymptomatic postoperatively.

following a persistent urinary fistula. A third failure occurred in a 71-year-old woman, with a large infected right hydronephrosis containing several stones and a functionless atrophic left kidney. At surgery a large branch of the renal artery was accidentally traumatized and was ligated. An infarct involving the lower pole developed. On the 11th postoperative day a massive hemorrhage occurred, and the splint and drainage tubes were removed. The patient recovered after repeated transfusions, but her infection and hydronephrosis persisted, although she was free from pain one year postoperatively, and the nonprotein nitrogen level was elevated to only 44 mg. per 100 cc.

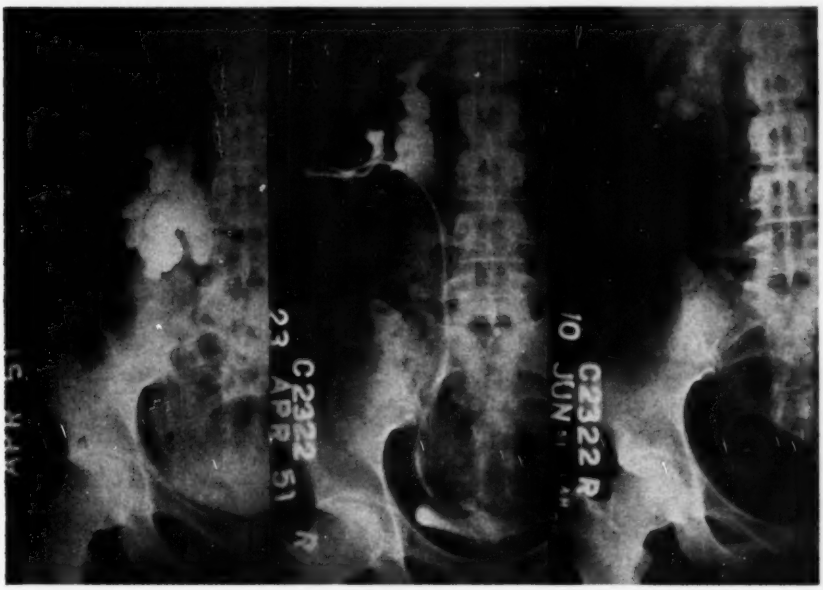

Fig. 8.—A white woman, aged 27, gave a history of sudden onset of radiating pain in the right upper quadrant, associated with nausea, vomiting, and total gross hematuria. Right kidney was explored April 5, 1951, with findings of a markedly distended pelvis and two aberrant vessels at the ureteropelvic junction, one crossing anteriorly and the other posteriorly, constricting the ureter. There was no intrinsic obstruction. The vessels were divided, a nephrostomy performed, and the ureter splinted for 18 days with a No. 14 F. polyethylene tube. The patient was asymptomatic postoperatively.

Four patients are listed as improved. These patients, although symptom-free and uninfected, showed only fair to little pyelographic improvement. In one of these patients (Fig. 9*A*) a stone later formed in the kidney operated on.

Operations were performed on three patients with solitary kidneys. The results of surgery are generally superior in these cases to those when a functioning mate is present. One of these cases was of great interest (Fig. 5*A*). The patient was a 28-year-old man, who had had a previous symphysiotomy and left nephrectomy for a horseshoe kidney. His right kidney had later become hydronephrotic owing to a ureteropelvic obstruction. An unsuccessful attempt was made elsewhere to repair this, and the patient was incapacitated by a persistent total urinary flank fistula. Another exploration was done, and the proximal ureter and pelvis were

Fig. 9.—*A*, a white man, aged 21, was admitted with a history of two recent attacks of left renal colic. Urologic evaluation revealed a ureteropelvic obstruction and a small amount of calculus in the upper third of the left ureter. The calculus passed spontaneously after indwelling ureteral catheterization for 24 hours. Exploration revealed a moderate hydronephrosis with extrinsic fibrous bands at the ureteropelvic junction and minimal intrinsic obstruction. The bands were freed. A nephrostomy tube and No. 14 F. polyethylene tube were brought through the renal parenchyma, and the splint pulled down the ureter and out the urethra. A Deming nephropexy was then done. The tubes were removed in 21 days. Three months postoperatively the patient returned with left renal colic, and x-rays revealed a small stone in the renal pelvis and marked pyuria. The results in this case must be considered poor.

B, a white man, aged 20, gave a history of pain in the left flank, associated with total gross hematuria of three weeks' duration. Operation revealed multiple, fine fibrous bands kinking the ureteropelvic junction, with moderately high take-off of ureter from the medial side of the pelvis. No aberrant vessels were present. Pelviureterolysis was performed with nephrostomy and ureteral splinting with No. 14 F. Levin tube for four weeks. The patient was asymptomatic postoperatively, with a follow-up of 18 months.

found to be densely involved in scar. The fistulous tract was followed down to a perforation in the renal pelvis. A pelviureterolysis was performed and a No. 14 Levin tube passed down the ureter into the bladder, the proximal end being brought out through a nephrostomy alongside a Foley drainage catheter. The splint was left in place for 49 days, and the patient made a prompt recovery. He was entirely well two and one-half years postoperatively. This case illustrates conclusively the value of ureteral splinting, because in no other way could a favorable result have been achieved.

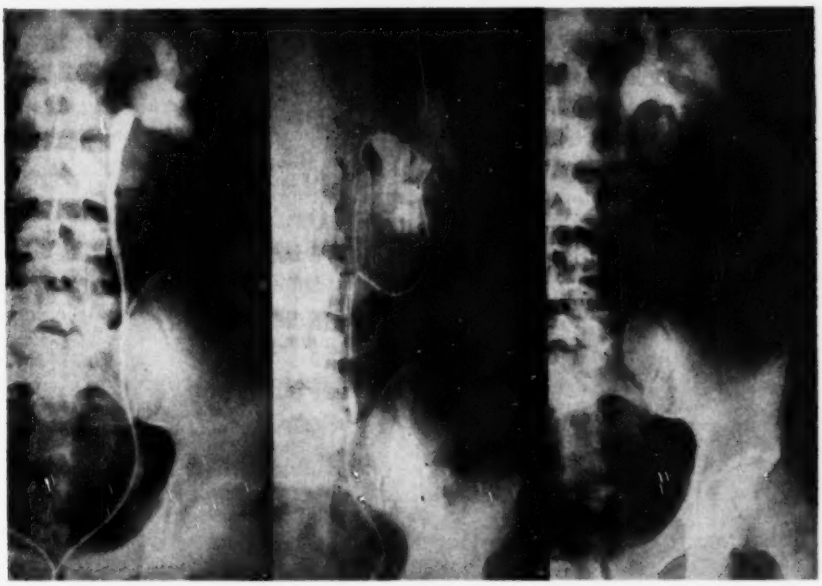

Fig. 10.—A white man, aged 26, gave a history of intermittent episodes of bilateral costovertebral angle pain lasting 2 to 3 days, of four years' duration. Urologic evaluation revealed chronic atrophic pyelonephritis on the right, and hydronephrosis due to ureteropelvic obstruction on the left. Left renal exploration revealed an intrinsic stenosis. A Davis-Rammstedt procedure was performed and the ureter splinted with a No. 12 F. T-tube for 21 days. The postoperative result was excellent. Six months later a right nephrectomy was performed.

SUMMARY

A study of 25 pyeloplastic procedures performed on 24 patients with ureteropelvic obstruction is presented.

There were no deaths in this series, and good results, meeting the criteria of symptomatic cure, freedom from infection and favorable functional and pyelographic findings, were obtained in 72% of the patients.

The management of this condition is reviewed and a plea for conservatism in the treatment of hydronephrosis is made. The keynote of treatment, we wish to emphasize, is simplicity; find the strictured area, incise it widely, and intubate with a minimum of suturing. Elaborate pyeloplasties are unnecessary and are less likely to be successful.

Several illustrative cases are also presented.

ACUTE NONSPECIFIC MESENTERIC LYMPHADENITIS

Possible Mechanism of Pain Illustrated by Two Cases

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HASTINGS, ENGLAND

ACUTE nonspecific mesenteric lymphadenitis is a common cause of pain in the abdomen of children. Notwithstanding Aird's [1] plea, made in 1945, for a wider recognition of this condition, it is still infrequently diagnosed; this is probably due to the fact that despite a typical clinical picture and a constant macroscopical pathology the etiology of the lesion is unknown.

Accurate diagnosis can be achieved in a large percentage of cases provided there is a clear conception of the condition. Aird [1] stated: "A considerable number of children in whom a diagnosis of non-specific adenitis has been made in the outpatient department were not admitted, but were confidently allowed to go home."

AGE INCIDENCE

Acute mesenteric lymphadenitis occurs most commonly between the ages of 3 and 15 years, with a peak incidence at both extremities of this age group. It is in the younger patients—3 to 6 years—that the greatest difficulty in distinguishing this condition from acute appendicitis occurs, and it is, therefore, in this group that laparotomy is most frequently performed. There should be no hesitation in operating if appendicitis cannot definitely be ruled out, as in this instance the interests of safety far outweigh those of accuracy of diagnosis.

CLINICAL FEATURES

The presenting symptom is abdominal pain, the older children generally giving a history of previous attacks. The pain of mesenteric adenitis is an acute central-abdominal colic; it causes crying, and the abdominal muscles may be held rigid, while the legs are sometimes drawn up onto the abdomen. "Between spasms of colic there is entire freedom from pain, and the patient is happier and livelier between spasms than is any patient who harbours an inflamed appendix" (Aird [1]). The colicky nature of the pain is emphasized by all writers and was found to be a prominent symptom in 10 of 13 cases reported by Adams and Olney.[2] Nausea is usually present, and vomiting occurs in about half the cases. Anorexia is common, but there is no distinctive alteration of bowel function.

1. Aird, I.: Acute Non-Specific Mesenteric Lymphadenitis, Brit. M. J. **2**:680-682 (Nov. 17) 1945.

2. Adams, W. E., and Olney, M. B.: Mesenteric Lymphadenitis and the Acute Abdomen: Report of 13 Cases, Ann. Surg. **107**:359-370 (March) 1938.

On examination the child is found to be flushed; the tongue is generally moist but has a slight white fur—it is considerably less dirty than it is found to be in cases of acute appendicitis. The patient is alert and restless, never prostrated by toxemia. The temperature is high and may be between 101 and 102 F. The pulse rate is increased, and in younger children 120 beats a minute is not unusual. A leucocytosis is invariably present for the first day or so of the illness. The child is rarely acutely ill for more than 48 hours.

Examination of the abdomen reveals tenderness over a localized area in the right lower quadrant. This area is higher than McBurney's point and more medial. "Shifting tenderness" was described by Klein [3] in 1938 and was said by him to be pathognomonic. When present it is suggestive of mesenteric adenitis, but it may also occur in appendicitis if the cecum is mobile. It may be absent in cases of mesenteric-gland involvement in which the cecum and the ascending colon are fixed and in cases in which the terminal ileum has a very short mesentery. "Shifting tenderness" is elicited as follows: The area of maximal tenderness is determined while the patient lies on his back; he then turns onto his left side, and on examination one minute later the site of maximal tenderness is found to have moved to the left of the umbilicus as the result of the pull of gravity on the bowel and its mesentery. I have seldom been able to elicit this sign with certainty.

The right rectus may exhibit an increase in tone, but rigidity has not been present in any patient I have examined. However, the patient's practitioner often mentions rigidity of the abdominal musculature in the communication which accompanies the child to hospital. This is an important point the significance of which will be discussed later.

Rectal examination is negative. It is essential to make this examination, as it has been found that inflammation of a pelvically situated appendix may produce signs and symptoms which closely simulate those of mesenteric adenitis. Differentiation lies in eliciting pelvic tenderness on rectal examination in cases of appendicitis.

In the acute form of mesenteric adenitis it is not usual to find enlarged glands in the axillae, the groins, or the triangles of the neck. There is no constant association with respiratory infection.

OPERATION FINDINGS

As most patients who come to operation do so because appendicitis cannot be excluded, an incision over the right iliac fossa is the usual one through which the limited laparotomy is performed. The first point which distinguishes mesenteric lymphadenitis from appendicitis is the absence of edema in the peritoneum of the anterior abdominal wall—a common finding in acute appendicular inflammation. On opening of the peritoneal cavity a small quantity of clear fluid escapes. Culture of this fluid is sterile.

When the appendix has been delivered it is usually found that the vessels on its serous surface are engorged, giving the organ a delicate pink blush. Nonspecific mesenteric lymphadenitis is no contraindication to appendicectomy, and this should be performed. The mucosa of the appendix exhibits signs of inflammation

3. Klein, W.: Nonspecific Mesenteric Adenitis: Report of 140 Cases, Arch. Surg. **36**:571-585 (April) 1938.

in proportion to the inflammatory state of other parts of the bowel; it may be slightly reddened and swollen, but it is rarely more severely inflamed. On histological examination in many cases it shows evidence of previous inflammation. Some authors cite this fact as indicating a connection between acute nonspecific mesenteric adenitis and appendicitis, but there is little other evidence in favor of this theory—enlarged mesenteric glands are seldom found in cases of frank acute appendicitis—and there is nothing to support the suggestion that appendicectomy is of definite value. On the contrary, Rosenburg [4] found that 19 of 29 patients with nonspecific mesenteric adenitis had further attacks of pain following appendicectomy. Adams and Olney [2] also reported pain after appendicectomy, and concluded that there is little evidence to support the opinion that appendicectomy is necessary to obtain a permanent cure. With this view Aird [1] concurred.

There is widespread enlargement of the lymph glands in the mesentery of the small intestine, and though it is not possible to say in our present state of ignorance concerning the etiology of the condition whether this most prominent feature is a primary or secondary pathology, it is this which gives the name to the syndrome. The glands lying in the ileocecal angle are usually maximally involved. The glands are enlarged, succulent and discrete; there is no evidence of perilymphadenitis. Histologically the lymph nodes present a nonspecific hyperemia and cellular hyperplasia. Culture of the glands is sterile, and animal inoculation of gland extracts has also failed to produce results. The vessels of the mesentery are engorged.

The terminal ileum is found to be completely relaxed and distended to the limits of normal. The wall is thin, and the bowel contents can be seen through it. The vascular pattern of the bowel wall is distinctly displayed. There may be mild hyperemia of the bowel wall, though frequently it is not reddened; its vessels are congested. Occasionally the terminal inch or two of the ileum is thickened by edema, presenting on palpation a sensation as of the pylorus. The distended condition of the terminal ileum is maintained for 2 or 3 ft. (61 to 91 cm.), and in this portion peristalsis is sluggish. As the ileum is traced proximally, the appearance changes and areas of tightly contracted empty bowel are found. Peristalsis in these areas is vigorous. Alternating areas of relaxed and contracted ileum are found.

The acute pyrexial phase of the illness is of short duration, usually 48 to 72 hours. Recovery is speedy, and the patient may be discharged from hospital as soon as the appendicectomy wound has healed. Patients treated without operation are fit for discharge in about five days.

Acute nonspecific mesenteric lymphadenitis is thus a clearly defined syndrome presenting a constant pathology. Its etiology is unknown. It is not a tuberculous condition; tuberculous mesenteric adenitis is a distinct lesion.

PAIN IN ACUTE NONSPECIFIC MESENTERIC LYMPHADENITIS

Klein [3] wrote: "The pain of mesenteric adenitis is not easily explained," while Foster [5] stated: "Some writers stress the sudden sticking pain, the variable vomiting, the duration of pain of only a few minutes and the freedom from pain between

4. Rosenburg, S.: Nonspecific Mesenteric Lymphadenitis: Report of 75 Cases, Arch Surg. **35**:1031-1044 (Dec.) 1937.

5. Foster, A. K., Jr.: Disease of Mesenteric Lymph Nodes: Its Relation to Appendicitis, Gastro-Intestinal Infections and Generalized Diseases; Report of 123 Cases; Possible Etiology and Treatment, Arch. Surg. **36**:28-52 (Jan.) 1938.

attacks." A history of severe pain causing violent crying is frequently obtained in cases of mesenteric adenitis, and, as mentioned earlier, the patient's practitioner commonly reports that muscular rigidity was present when he examined the child, though as a rule there is no evidence of this by the time the patient has arrived in hospital.

The pain in this condition is, then, a severe intestinal colic. Enlargement of the mesenteric glands of itself will not produce this pain, and a further cause must, therefore, be present.

Acute nonspecific mesenteric adenitis is frequently diagnosed in the Royal East Sussex Hospital, and the patient is then treated without operation. I have recently, however, operated on three patients in the 3- to 6-year age group in whom acute appendicitis could not be excluded with complete confidence. All three presented at operation the typical picture of mesenteric adenitis without acute inflammation of the appendix. Appendicectomy was performed in each case.

In two of the three cases, however, in addition to the typical macroscopical pathology heretofore detailed, there was found in an area of contracted ileum a small ileoileal intussusception.

REPORT OF CASES

CASE 1.—A 5½-year-old girl was admitted with a history of pain in the right iliac fossa and umbilical region of three days' duration. The pain had become more severe on the day of admission, was colicky in nature, and had caused the child to draw up her knees. She was constipated and had lost her appetite. There had been no vomiting. Temperature was 100 F. and pulse rate 130. The tongue was clean. There was tenderness in the right iliac fossa, with guarding of the lower half of the right rectus. At laparotomy, through a transverse incision over the right iliac fossa, typical mesenteric adenitis as previously described was found, plus an ileoileal intussusception 2 in. (5 cm.) in extent and situated about 3 ft. (91 cm.) from the ileocecal angle. This reduced easily.

CASE 2.—A boy of 3 years and 11 months had had abdominal pain for three days associated with loss of appetite. There had been no vomiting, but he had felt sick. Temperature was 101.5 F. and pulse rate 125. On examination he was found to be tender in the right iliac fossa. There was no rigidity of the abdominal musculature and no guarding, yet the patient's doctor stated that the abdomen had been very rigid when he had examined it. At laparotomy through an incision similar to that used in Case 1, typical mesenteric adenitis was found, and in addition there were an ileoileal intussusception 3 in. (7.6 cm.) in extent and a second area at which intussusception was commencing. The intussusception reduced readily. The fluid in the peritoneal cavity was cultured but gave no growth.

It is worth noting that in Case 2 the intussusception was found high up the ileum and would have been overlooked in a routine search of the lowest few feet of the small bowel for a Meckel diverticulum. My attention having been drawn to the possibility of the presence of an intussusception by the previous case encouraged me to trace the ileum higher than I would otherwise have done. A high location may help to explain why this feature has not been noted previously, and, indeed, Adams and Olney,[2] reporting on the eight cases in which they operated as for acute appendicitis, stated: "Exploration was limited in our operated cases. . . . Thus the findings are limited to a local area."

It will be readily appreciated that the common description of the pain which occurs in mesenteric adenitis as "colicky," "severe," and "of short duration," together with the "freedom from pain between attacks," is adequate to describe the pain of intussusception. Similarly, the drawn-up knees and the rigid abdominal

musculature sometimes found are typical features of intussusception. Thus it appears that the spasms of pain which occur in patients suffering from mesenteric adenitis may be due to small ileoileal intussusceptions which almost invariably reduce themselves and give rise to no more serious consequences. On this basis the discrepancy between the rigid abdominal wall found by the patient's doctor and the relaxed musculature found when the patient is examined in hospital is adequately explained; the practitioner sees the child during a phase of intussusception which has reduced itself before the patient's arrival in hospital. It follows that this apparent discrepancy may be of some importance in diagnosis.

With reference to this postulate as to the cause of pain in mesenteric lymphadenitis, there is a significant reference in the literature. Ireland,[6] in 1938, giving an account of 22 cases of this condition, stated: "Appendectomy was performed in all but two. The appendixes were not removed from these two because intussusception was present in one and intestinal hemorrhages had preceded operation in the other." Appendicectomy being regarded as contraindicated, one assumes that the intussusception found was of the ileocolic variety, but in view of the findings reported herein, it is not unreasonable to suggest that it had commenced as an ileoileal intussusception, which, having failed to reduce spontaneously, had progressed to the more advanced stage of ileocolic intussusception.

ILEOILEAL INTUSSUSCEPTION

Maingot,[7] discussing small-bowel intussusception, wrote: "Enteric—Here the small gut becomes invaginated into the small gut (jejuno-jejunal or ileo-ileal). This is a rare type, accounting for perhaps about ten per cent of cases in adults. It is seldom encountered in infants." Yet, reported here are two instances found in two successive operations on patients with typical mesenteric adenitis, each patient's history containing a statement fully in accord with the finding of intussusception though completely characteristic of mesenteric adenitis. In view of the general agreement on the colicky nature of the pain which occurs in mesenteric adenitis and the finding of this rare type of intussusception in typical examples of this condition, it is not unreasonable to postulate a definite relationship. Adams and Olney[2] in their account of 13 cases published in 1938 drew attention to the fact that "the colicky, intermittent character of the abdominal pain" indicated that small-bowel dysfunction played a prominent part in the syndrome, and they went on to say that intussusception must be considered in the differential diagnosis. As stated, these writers admitted that their laparotomies were limited in extent, and this fact may be the reason why the findings herein reported were not discovered by them.

Intussusception may follow an upset of normal peristaltic rhythm and has certainly been found at laparotomy presumably owing to the stimulation of handling of the bowel. Personally I have not been able to invoke intussusception by the normal slight traumata to which the intestine may be subjected at laparotomy and have not previously found it at operation. Agonal intussusceptions are well known to pathologists and are due to abnormal peristalsis immediately ante or post mortem. There

6. Ireland, J.: Etiologic Factors of Mesenteric Lymphadenitis, Arch. Surg. **36**:292-302 (Feb.) 1938.

7. Maingot, R.: Abdominal Operations, Ed. 2, London, H. K. Lewis & Co., Ltd., 1948, p. 950.

is no reason to suppose that abnormal peristalsis does not occur in mesenteric adenitis, and Rosenburg [4] in discussing the colicky nature of the pain suggested that it is due to intestinal spasm secondary to irritation in the mesenteric root. I consider it likely that the spasm proceeds to small-bowel intussusceptions.

A case was reported by Gill,[8] in 1938, of multiple ileoileal intussusceptions in a child following the taking of a purgative. He added, "The only other finding of note was the presence of large matted glands in the mesentery opposite the affected area of intestine, and these were thought to be tuberculous." The diagnosis of the matted glands as tuberculous is certainly correct, as in nonspecific mesenteric adenitis the absence of perilymphadenitis is a notable feature.

MacKeith and O'Neill,[9] recently discussing recurrent abdominal pain in children, wrote, "The attribution of recurrent abdominal pain to chronic nonspecific adenitis is of interest," and they went on to say, "Possibly these descriptions of non-specific mesenteric adenitis as a common cause of abdominal pain in childhood are an example of medical mythology in the making." Two cases are not sufficient basis for dogmatic statements. It is hoped that this report will stimulate others to seek enteric intussusceptions when they encounter nonspecific adenitis at operation and thus show that there is a definite cause of pain in this condition and that it is unnecessary to invoke, as MacKeith and O'Neill [9] did, the diagnosis of "disharmonious mental states" to explain it.

SUMMARY

Acute nonspecific mesenteric adenitis is a common cause of abdominal pain, colicky in nature, in children. The clinical features and the pathology are described.

An account is given of two patients operated on because acute appendicitis could not definitely be excluded. In addition to the typical features of mesenteric adenitis, an ileoileal intussusception was found in each.

It is suggested that ileoileal intussusception may be the cause of the colicky pain of mesenteric adenitis.

8. Gill, W. G.: Multiple Intussusceptions in a Child, Brit. J. Surg. **25**:707 (Jan.) 1938.

9. MacKeith, R., and O'Neill, D.: Recurrent Abdominal Pain in Children, Lancet **2**:278-282 (Aug. 18) 1951.

Case Reports

MASSIVE HEMORRHAGE FROM MALIGNANT HEPATOMA

Report of a Case with Subsequent Transdiaphragmatic Resection

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MASSIVE intraperitoneal hemorrhage from liver tumors has been reported previously. Thirteen cases have been found in the literature by Specter and Chodoff,[1] including one of their own. The relative rarity of hepatoma in the Caucasian race and this unusual manifestation of it results in nearly every case in the diagnosis not being made until exploratory laparotomy or at autopsy. From the available information in the reported cases it is evident that very few of the patients reach the operating room in time for a successful outcome. Six patients died suddenly, and the diagnosis was established at autopsy; four patients underwent exploration but did not survive, and only three patients survived the initial episode, including Specter and Chodoff's patient who had had a resection at the initial operation.

Every surgeon is familiar with the clinical picture of acute peritoneal irritation produced by fresh intraperitoneal bleeding, but this situation is most commonly encountered in the female pelvis. The following case represents an example of intraperitoneal hemorrhage which was, in every respect, indistinguishable from perforated peptic ulcer and which is of additional interest because of its location and the technical problems of subsequent resection.

REPORT OF A CASE

S. H. G. G., a 38-year-old Chinese man, entered the hospital with a complaint of sudden onset of severe pain in the right upper abdominal quadrant four hours before admission. The pain was moderate at first and gradually became extremely severe. It was later referred to his right shoulder. He denied vomiting and denied history of indigestion, bowel irregularity, or melena. The patient reported that two days before admission he had had a similar right upper quadrant pain of less severity which lasted about two hours and disappeared. The past history was not remarkable. He denied serious illnesses, injuries, or previous operations. He had no known weight loss, malaise or fever, cough, hemoptysis, chest pain, or dyspnea. Physical examination revealed a well-developed, young-appearing man apparently in good health but in obvious acute abdominal distress. The skin was pale, moist, and cool. The blood pressure was 110/90 and the pulse rate regular. The chest was clear to percussion and auscultation, and the heart size was normal. There was a voluntary spasm throughout the abdomen with a moderately acute spasm in the right upper quadrant. Cough and rebound tenderness were referred to the right upper quadrant. No peristaltic sounds could be heard. Laboratory studies showed a hemoglobin of 19.0 gm. per 100 cc. (repeated) and a white blood cell count of 18,100. The urine had a pH of 6.0 and a specific gravity of 1.015, with a 1+ reaction for albumin and no sugar. The urine sediment showed 3 to 5 white blood cells per high power field.

Dr. Roe is Assistant Clinical Professor of Surgery, University of California School of Medicine, San Francisco; formerly Resident Surgeon, Massachusetts General Hospital, Boston.

1. Specter, J., and Chodoff, R. J.: Massive Intraperitoneal Hemorrhage from Carcinoma of the Liver: Review of Literature and Case Report, Surgery **27**:457, 1950.

A clinical diagnosis of perforated ulcer was made and the patient sent to the operating room. The absence of previous digestive symptoms is fairly common in perforated ulcer and did not cause us to hesitate in this case. The sudden onset, localization of signs, and leucocytosis were considered sufficient indication for immediate laparotomy, and an abdominal x-ray film for subdiaphragmatic air was not considered necessary. Perforated ulcer is commonly associated with shock-like signs of pallor, sweating and anxious facies without a significant drop in blood pressure. His age, constant weight, high hemoglobin value, and lack of previous symptoms were against serious consideration of ruptured carcinoma.

Champeau and Ferry [2] have described in the French literature a syndrome of "pseudo perforation" with similar signs and symptoms in four cases of liver tumor, but in each case the surgical exploration failed to demonstrate blood. In previously mentioned cases with bleeding, of course, the clinical picture was not uncommonly that of upper abdominal peritonitis, though the associated shock and collapse were more evident than in our case.

Laparotomy revealed a large amount of dark blood in the peritoneal cavity; 1,500 cc. was evacuated, and a necrotic tumor at the dome of the right lobe of the liver was discovered to be bleeding actively. A biopsy was done and the area covered with oxidized (oxycel®) gauze, which

Fig. 1.—External surface of resected specimen showing original sutures. Superficial fibrin, clot, and oxidized gauze have been removed. Dotted line shows approximate area of additional resection.

was secured with mattress sutures. Exploration of the abdomen failed to demonstrate any other abnormality or tumor.

Postoperatively his recovery was uneventful. The pathologist returned a report of hepatoma. During the postoperative course extensive x-ray studies were made to demonstrate metastatic disease, but none was recognized. Nine days after the initial operation he underwent a second procedure for resection of the tumor.

The following operative procedure was carried out. With use of intratracheal gas, oxygen, and ether anesthesia, a thoracoabdominal incision was made through the bed of the ninth rib. The diaphragm was opened, and the liver was mobilized by dividing the triangular suspensory ligament and the round ligament so as to permit approaching every aspect. A fibrin clot-covered area obscured the previously described tumor in the dome of the right lobe of the liver and extended somewhat medially. The absorable surgical (gut) sutures and the oxidized gauze were essentially intact. The tumor measured approximately 3 by 4 in. (7.5 by 10 cm.) on the surface

2. Champeau, and Ferry: Syndromes psuedo-perforatifs par cancer du foie, Arch. mal. app. digestif **38**:799, 1949.

and was perhaps 2 in. (5 cm.) deep. Careful inspection failed to demonstrate disease elsewhere in the liver. The left lobe was small and the cleft not well defined so that a total right lobectomy did not seem advisable. The portal vein, the right hepatic artery, and the hepatic vein from the right lobe were dissected out and prepared for temporary occlusion with a noncrushing clamp. An ellipse of overlapping, multiple, deep circular sutures of No. 1 chromic gut were placed around the tumor with a liver needle and ligated, leaving a margin of about 2 cm. in every direction. Initial attempts were made to excise the tumor with electrocautery, but excessive bleeding made it necessary to occlude the previously mentioned vessels. Even then the field was far from dry, and a small segment of tumor was inadvertently transsected on the medial end. This area was secondarily excised with an adequate margin. No gross tumor was left behind.

Gut mattress sutures were used to close the several large gaping vessels and bile ducts on the cut surfaces, and the teacup-sized defect in the dome of the liver was partially closed with deep circular sutures. The occluded vessels were then unclamped, having been closed for 14 minutes,

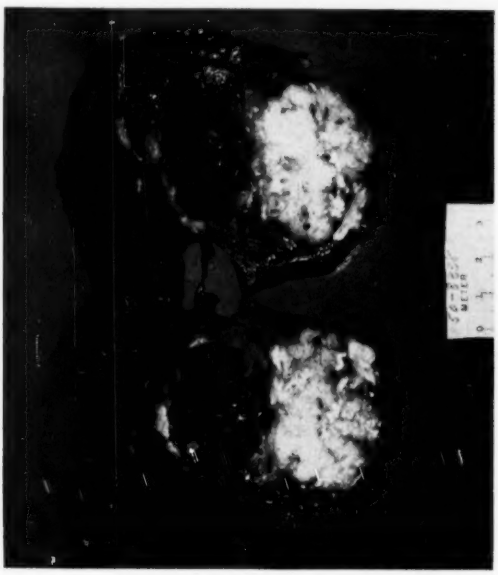

Fig. 2.—Cut surface of specimen in transverse plane, showing area of hemorrhage in part of tumor.

and the liver returned to good color, except for a small area which remained dull grey but did not seem to be grossly infarcted. Neither blood nor bile was seen to be leaking from the defect.

The subdiaphragmatic space was drained and the incision closed. During the procedure the patient received 5 pt. (2,500 cc.) of blood, which probably more than replaced his loss. His blood pressure and general condition remained excellent throughout.

His postoperative course was complicated by a bloody discharge through the drainage site. The foul discharge was evacuated on the 11th postoperative day. He gradually improved and was discharged home on the 21st day. A sulfobromophthalein test on the second postoperative day showed 46% retention of dye. Studies made on the 15th postoperative day gave the following results: alkaline phosphatase, 8.1 units per 100 cc.; sulfobromophthalein retention, 16%; 0.3 mg. of bilirubin per 100 cc. (direct van den Bergh reaction) and 0.5 mg. total bilirubin per 100 cc.; cephalin flocculation, 1 + at 48 hours; thymol flocculation negative; serum albumin, 3.70 gm. per 100 cc.; globulin, 2.54 gm. per 100 cc., and albumin-globulin ratio, 1.5.

He continued to have intermittent bouts of fever and malaise until he was readmitted and a subphrenic abscess drained, seven weeks after discharge. Following this his recovery was uneventful. A follow-up one year after operation revealed no clinical evidence of recurrence, no complaints, and a weight gain of 12 lb. (5.4 kg.). A follow-up report at the end of two years revealed that he was alive and healthy and had maintained his weight. He was reported to have undergone a laparotomy for small bowel adhesions on Aug. 30, 1952, at which time there was no evidence of intra-abdominal malignancy.

COMMENT

Partial liver lobectomies and total left lobectomies for primary and metastatic tumors have been reported by many authors. The feasibility and technique of the procedure are well established today for transabdominal resection of wedge segments or pedunculated tumors. Masses of remarkable size have been resected from the liver,[3] the largest of which was reported in 1921 by W. Wendel,[4] a German surgeon who removed a 940 gm. adenocarcinoma involving most of the right lobe. In our case, however, the technical aspects of removing a relatively small tumor from the dome of the right lobe presented a somewhat different problem. At the time we were unable to find any reports of successful total right lobectomies and of course were reluctant to sacrifice such a large amount of normal liver tissue.

This approach to partial liver resection will probably have limited application, since most lesions can be removed simply with a wedge taken from the liver edge. Our experience would suggest, however, that part or all of this organ can be temporarily isolated from the circulation with safety and that the transthoracic approach makes technically feasible resections which would otherwise be hazardous or impossible.

3. Chavache, H.: Primary Carcinoma of the Liver, Am. J. Surg. **43**:96, 1939.
4. Wendel, W.: Ueber Leberlappenresektion, Arch. Klin. Chir. **114**:982, 1920.

A PLASTIC PROSTHESIS FOR RESECTED ESOPHAGUS

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ENCOURAGED by the experimental work on the replacement of portions of the esophagus by a plastic tube in dogs,[1] it was thought feasible to attempt this procedure in an elderly patient with a carcinoma of the lower end of the esophagus with metastasis. The patient was an extremely poor risk, and it was our judgment that it would have been more dangerous and time consuming to attempt the more conventional method.

REPORT OF A CASE

A. G., a 75-year-old white man, was admitted to the Sinai Hospital in October, 1950, with a complaint of having difficulty in swallowing for one week.

The past history was noncontributory.

The family history revealed that the father died of cancer of the "neck."

An interview with the patient dated back to only a week or so prior to admission, when he stated that on attempting to swallow some meat, "it stuck in my chest." He was able to get this down with copious amounts of water. However, since then he had had difficulty in swallowing and regurgitated all solids and most liquids. He had some pain in the lower portion of the chest when attempting to swallow foods of solid or semisolid consistency.

On physical examination the patient showed some evidence of weight loss, but otherwise the examination revealed no abnormalities. The blood work-up was normal.

Esophagoscopy revealed no abnormalities. An esophagram with fluoroscopy revealed a smooth, persistent pressure defect in the lower esophagus measuring 3.5 cm. in length. An intramural submucous lesion was postulated (Fig. 1).

Operation was performed on Nov. 10, 1950, with use of thiopental (pentothal®) sodium and intratracheal cyclopropane ether anesthesia. A thoracotomy was done, resecting the left eighth rib from the anterior axillary line to the vertebral border. The lung was packed off; the pulmonary ligament was clamped, cut, and ligated, and the mediastinal pleura was incised over the lower esophagus. A walnut-sized esophageal mass was palpated about 3 cm. above the cardia. A mediastinal gland was excised, and the frozen section revealed carcinoma. Glands were also palpated along the left gastric artery. The esophagus was then dissected free from its bed, and by blunt-finger dissection the hiatus was enlarged and a portion of the cardia of the stomach was pulled through. The esophagus was then cut across approximately 4 cm. proximal to the lesion, and distally the stomach was cut across below the tumor. The edges of both the esophagus and the stomach were grasped with Babcock clamps. A plastic tube 12 cm. in length and 1.2 cm. in diameter was anastomosed proximally to the esophageal stump and distally to the stomach in the manner previously described in the experimental work. This anastomosis took approximately 15 minutes to complete. The deficit bridged was about 8 cm. in length. The mediastinal pleura was closed tightly with interrupted nonabsorbable surgical (silk) sutures and the diaphragm attached to the portion of the stomach brought through the hiatus. The lung was reexpanded and the chest closed. Tube drainage was established one interspace below the initial incision. The patient maintained a normal pulse rate and normal blood pressure throughout the procedure, and only 750 cc. of blood was given during the operation.

From the Department of Surgery, Sinai Hospital.

1. Berman, E. F.: The Experimental Replacement of Portions of the Esophagus by a Plastic Tube, Ann. Surg. **135**:338 (March) 1952.

Fig. 1.—Roentgenogram taken after preoperative barium swallow, showing a defect in the lower end of the esophagus approximately 3.5 cm. in length.

Fig. 2.—Roentgenogram taken after barium swallow four weeks after operation, showing the normal esophagus with the barium flowing readily through the plastic tube, which has replaced the lower 8 cm. of esophagus, into the stomach.

The pathologic diagnosis was adenocarcinoma of the lower end of the esophagus.

After operation, a Levine tube was kept in the esophagus proximal to the plastic tube for three days, with continuous suction. Penicillin and streptomycin were administered, and fluids were given intravenously. On the third postoperative day the Levine tube was removed and the patient was given a full liquid diet. He had a low-grade fever for about three weeks, which gradually subsided. His diet was gradually supplemented until he was eating normally by the time of discharge. He was admonished to chew his food well.

Four weeks after operation a barium swallow revealed the barium going through the proximal esophagus and through the plastic tube into the stomach, with no obstruction (Fig. 2).

The patient convalesced nicely at home, with no physical incapacity and no difficulty whatsoever in swallowing any type of food. In February, 1951, he was readmitted with pneumonitis

Fig. 3.—Roentgenogram done eight weeks after operation, showing the continuity of the barium after the tube was removed by esophagoscopy. At the upper edge there is some evidence of constriction, but the patient had no difficulty in swallowing and was able to eat normally until he died.

at the base of the left lung but was discharged after being given antibiotics for a few days. He was perfectly well until June, 1951, when he returned with obvious metastasis, as evidenced by weight loss, abdominal pain, a palpable liver, and a distended abdomen. At this time it was decided to attempt the removal of the tube. As was shown in the experimental animal, an epithelium-lined fibrous sheath forms around the tube, and, after removal of the tube by esophagoscopy, the animal eats normally with no evidence of stricture formation. With the patient under thiopental anesthesia, the esophagoscope was placed down to the upper rim of the tube. The rim of the tube was grasped with a long forceps and the tube, esophagoscope, and forceps pulled out together. Some force was necessary to do this. The patient ate one day after the tube was removed, and one month later an esophagram was taken (Fig. 3), showing the barium

being propelled through the esophagus and new sheath into the stomach. There was one area which seemed to be constricted. The patient ate normally after this for approximately two months until he died quietly in August, 1951. No permission for autopsy was obtained.

COMMENT

The results obtained in this first patient, in whom an esophageal deficit was bridged by a plastic tube, has borne out some of the hypotheses arrived at from the experimental work.

1. There was a minimum of operative trauma, and the operating time was short.

2. There was no associated shock, and a relatively small amount of blood replacement was necessary.

3. There were no time-consuming or technical difficulties in anastomosing the tube to either end of the esophagus.

4. The postoperative course was relatively smooth. The patient ate a normal diet on discharge, with no discomfort.

5. Convalescence was uneventful, and even with obvious regional metastasis there was no evidence of obstruction at or near the anastomosis. It was found that the tube could be removed by esophagoscopy after eight months and the patient would still have a functional esophagus. (The tube may remain in indefinitely, if desired.)

SUMMARY

A resected portion of the esophagus containing a carcinoma was replaced by a plastic tube which maintained continuity of this structure with the stomach. Eight months after operation the tube was removed by esophagoscopy, leaving a functional epithelialized fibrous sheath to maintain continuity. The patient lived for 10 months in comfort, finally dying of metastasis.

ABDOMINAL ANEURYSM WRAPPING WITH POLYVINYL FORMAL SPONGE

Report of a Case with Duodenal Perforation

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SANTA ROSA, CALIF.
RICHARD L. RAPPORT, M.D.
FLINT, MICH.
AND
GEORGE M. CURTIS, M.D.
COLUMBUS, OHIO

IN A RECENT report, Grindlay and Waugh [1] suggested the use of polyvinyl-formal plastic sponge [2] to reinforce abdominal aortic aneurysms and reported its use in four cases. Since one of us had been working both experimentally and clinically with this material in thoracic plombage,[3] we were acquainted with its chemical and physical properties, as well as tissue response to its implantation. It appeared to us that there was considerable merit in this conception of its use. Our opinion was based primarily on the following two factors: (1) the moistened sponge is soft and would not be likely to effect a pressure necrosis of the aneurysm wall, and (2) the sponge, though generally inert, produces a low-grade foreign body response in tissues, consisting essentially of granulation tissue with an occasional foreign body giant cell. Accordingly, in view of the generally unsatisfactory nature of available therapeutic measures for this condition, we felt clinical trial was not only justifiable but desirable, and when the patient whose case report follows was referred to us, we felt no hesitation in suggesting the procedure as a reasonably safe, though experimental, method of treatment.

REPORT OF CASE

History and Physical Examination.—J. G., a 50-year-old white man, a machinist, entered Ohio State University Hospital for his first and only admission on Oct. 11, 1951. His illness began approximately 15 years previously, at which time in his occupation as a machinist he started to operate a drill press as his daily work. In doing this he utilized his epigastrium to push large metal objects into position and maintain them there for drilling. Shortly thereafter he noted the onset of a periodic epigastric "jerking with each heart beat" and recurrent attacks of

From the Department of Surgical Research, Ohio State University, College of Medicine.

1. Grindlay, J. H., and Waugh, J. M.: Plastic Sponge Which Acts as a Framework for Living Tissue: Experimental Studies and Preliminary Report of Use to Reinforce Abdominal Aneurysms, A. M. A. Arch. Surg. **63**:288-297 (Sept.) 1951.

2. Ivalon® sponge is manufactured by Ivano Inc., 123 E. 21st St., Chicago 16.

3. Thurlow, A. A., Jr.: Extrapleural Pneumonolysis with Plombage in Pulmonary Tuberculosis, Thesis, Ohio State University, 1952.

precordial pain, which had continued to the present admission. He saw his physician, who diagnosed the condition as angina pectoris. Ten years previously the presence of a palpable pulsating mass was noted by the patient for the first time. Five years later he had his most serious episode, with radiation of sudden severe pain from his lower midchest down into his anus. An electrocardiogram taken at that time was considered to be normal. With subsequent attacks, pain radiated into the lumbar area. During the 18 months prior to entry the patient had become progressively worse, with increasing dyspnea, easy fatigability, backache, and anorexia. During the immediate three weeks prior to admission, epigastric, back, and chest pains were constant. The epigastric mass increased in size notably and for the first time was felt by his physician. Digitalis was prescribed shortly before entry but had no significant effect.

On physical examination the patient appeared poorly nourished and chronically ill. Emphysematous breath sounds with occasional subcrepitant rales were heard at both bases, but otherwise the lungs were clear. Cardiac examination was normal; the blood pressure was 130/80.

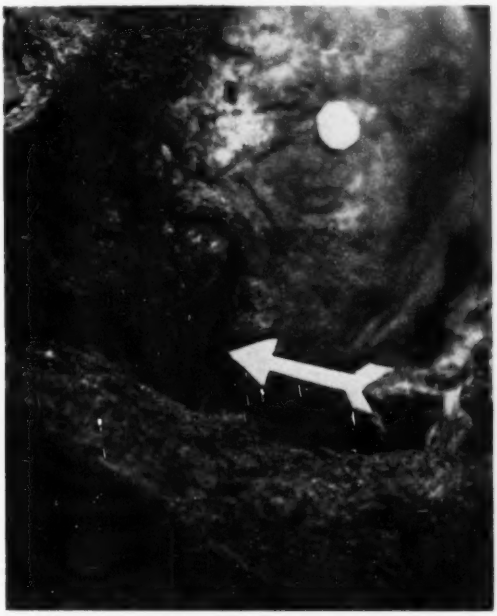

Fig. 1.—Photograph of fixed postmortem specimen, showing left lateral wall of aneurysm (dot) with site of rupture (arrow). It is to be noted that rent stops anteriorly at site of sponge attachment, which is seen in upper left corner.

Abdominal examination showed the liver to be enlarged 2 cm. below the costal margin. In the midline, extending from the epigastrium to below the umbilicus, was a large fusiform, pulsating, expansile, fixed mass. This was moderately tender to palpation. All extremities showed normal pulses and blood pressures.

Laboratory Data.—Urinalysis, serology, blood counts, and blood chemistries were within normal limits. X-rays and fluoroscopy showed slight pulmonary emphysema and bilateral renal enlargement. A mass was noted behind the shelving portion of the right lobe of the liver, displacing the stomach and duodenum to the left and the duodenal bulb, as well as the left ureter, anteriorly. Expansile pulsation of the abdominal aorta was seen. The electrocardiogram showed somewhat low voltage in limb leads and a vertical electrical axis but no definite abnormality. A retrograde aortogram revealed aneurysmal enlargement of the entire abdominal aorta.

Fig. 2.—Photograph of aneurysm on midline section, showing it filled with laminated clot and sponge attachment anteriorly. Dot indicates luminal side of rupture.

Fig. 3.—Photograph of postmortem fixed specimen, showing third portion of duodenum, which has been opened anteriorly, being eroded by the sponge from behind (large arrow). Small arrow indicates a second site of duodenal perforation; square indicates the pancreas.

Hospital Course.—With the establishment of a definite diagnosis, polyvinyl (ivalon®) sponge wrapping was considered and judged to be indicated. The sponge was prepared by gelatin-freezing fixation and slicing, as described by Grindlay and Waugh.[1] Slices were approximately 3 to 4 mm. by 14 cm. by 8 cm. Sponge sterilization was effected by continuous tap-water washing for 48 hours, multiple distilled-water rinsings, and, finally, immersion for 48 hours in 1:1000 benzalkonium (zephiran®) chloride solution. The operative procedure was done on Oct. 17, 1951, via a left paramedian incision. Exploration revealed a "large fusiform abdominal aortic aneurysm extending from 2 cm. below the diaphragm to the bifurcation." The transverse colon was pushed inferiorly to the level of the aortic bifurcation, and the third portion of the duodenum was stretched and displaced anteriorly by the mass. The posterior parietal peritoneum

Fig. 4.—Photomicrograph showing site of sponge attachment to aneurysm wall. Clear spaces represent location of sponge material which has retracted, for most part, during processing. Surrounding these spaces is granulation tissue (with foreign body giant cells, mononuclear cells, fibroblasts, and capillaries) which filled the sponge vacuoles. Aneurysm wall shows considerable acute and chronic inflammatory cellular infiltration.

was incised and the retroperitoneal space entered. By blunt and sharp dissection the aneurysm was exposed around to the vertebral bodies on either side. The wall was relatively smooth and no branches were noted to arise from its anterior and lateral surfaces. The sponge strips were placed over the aneurysm so that, with the vertebral bodies posteriorly, complete encasement was effected. No sutures were placed to secure the sponge strips because it was felt that the relatively thick and tight-fitting sheath of periaortic tissues and peritoneum would effectively prevent movement. Closure of the sheath was done with interrupted nonabsorbable surgical (silk) sutures after 200,000 units of penicillin and 0.5 gm. of streptomycin were placed in the space.

Postoperatively the patient's course was complicated by epigastric pain and fever, ranging up to 101 F. despite adequate daily administration of penicillin and streptomycin. He gradually improved after ambulation and catharsis, with evacuation of considerable amounts of retained barium, and was discharged on the 10th postoperative day. At this time, though the pulsating mass still was present and his temperature was 99 F., he was eating fairly well; the abdomen was soft, bowel sounds were normal, daily soft stools were present, epigastric pain was minimal, and his wound was well healed.

Course Following Discharge.—Three days following discharge he was seen by his physician, who, because of a temperature of 101 F. and abdominal pain, gave him penicillin and sulfamerazine. His temperature returned to normal after a few days and, gradually, over a two-week period, the abdominal pain disappeared. The patient felt quite well for the next six weeks and then there occurred, with relatively sudden onset, severe nausea and vomiting together with fever and upper abdominal and back pain. He was hospitalized at his community hospital, where intravenous pyelograms revealed obstruction of the right ureter. Cystoscopy with catheterization of the right ureter and renal pelvis was done, with drainage of purulent urine. After this, his temperature returned to normal and he noted considerable pain relief. Some epigastric distress and backache remained, however. A gastrointestinal series was done several days later and showed extravasation of barium from the distal duodenum into the perianeurysmal space. Despite this, the patient's improved status continued until Jan. 9, 1952, at which time he suddenly felt faint and shortly thereafter vomited a large amount of dark blood. Hematemesis continued periodically associated with recurrent states of shock (in spite of transfusions) until approximately 48 hours later, when he died suddenly, with a massive evacuation of old and new blood per rectum.

Postmortem Examination.—Autopsy at the local hospital revealed that the stomach and entire intestinal tract were filled with blood. The aneurysm had ruptured in its left posterolateral aspect, there being a 2.5 cm. rent in its wall (Fig. 1). There was a fairly large, thick, fibrous-walled space about it filled with blood and loose-lying sponge slices. Over a small area, anteriorly, sponge was attached by "fibrous" union to the aneurysm wall (Fig. 2). There were four perforations in the posterior wall of the duodenum. Presumably these were due to a pressure necrosis by the sponge, several slices of which had migrated and collected retroduodenally (Fig. 3). They allowed free communication between the duodenal lumen and the periaortic space. Obstruction of the right ureter by pressure from the sponge was evident also. The aneurysm measured 10 by 15 by 10 cm. On the cut section its wall varied in thickness from 3 to 7 mm. It was filled with laminated clot (Fig. 2). Grossly, no changes in the sponge material were evident. The essential specimen was mailed to the Ohio State University Hospital for further study, and the above findings were confirmed. Multiple microscopic sections were made, which were compatible with the interpretation of the gross findings (Fig. 4).

COMMENT

To our knowledge at the time of writing (May 1, 1952) no end-results from the treatment of abdominal aneurysms by wrapping with polyvinyl (ivalon®) sponge have been reported. In view of the excellent clinical follow-up, the postmortem studies, and the instructive features of this case, it appears worthy of record. The following two significant features should be emphasized:

1. Sponge slices should be fixed in position by sutures. Certainly in this case failure to suture allowed migration of sponge slices and their collection retroduodenally, producing the ureteral obstruction and the duodenal perforations. Other possible causes of the perforations, such as stretching and displacement of the duodenum by the aneurysm, the retroperitoneal operative dissection, affinity of the sponge for water and subsequent fluid accumulation, etc., were considered but on the basis of the findings did not appear to be tenable.

2. Active reinforcement of the aneurysm wall against further thinning and rupture does appear to be possible, as evidenced by the actual attachment which

was noted. Of course, to be worth while, union must be uniformly present over the aneurysm. The absence of uniform fusion in this instance is attributable to the sponge migration and duodenal perforation.

SUMMARY AND CONCLUSIONS

A case report is presented in which an abdominal aortic aneurysm was treated by wrapping with polyvinyl-formal sponge slices which were not sutured into position. The postoperative course was complicated by fever and abdominal pain which subsided, giving the patient a short period of symptomatic relief. Approximately two months after operation there occurred perforation of the duodenum at several points, obstruction of the right ureter with a hydronephrosis, and finally rupture of the aneurysm. Postmortem examination revealed that the sponge slices had migrated and collected retroduodenally, producing four perforations in the posterior duodenum by pressure necrosis.

The following two conclusions are apparent: 1. The demonstration of actual union between aneurysm and sponge indicates that effective reinforcement may be possible by this method. 2. It is essential that sponge slices used for wrapping be sutured into position.

FOREIGN BODY OF EMBOLIC ORIGIN IN RIGHT VENTRICLE OF HEART REMOVED WITH SUCCESSFUL RESULT

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A FOREIGN body brought to the heart through the major vessels is a rare condition, encountered, as a rule, only under wartime conditions, when multiple small foreign bodies have opportunity to penetrate the human body. It would be impossible to estimate the number of foreign bodies which have penetrated large venous channels and, at the same time, closed over this wound of entrance. Later, if the foreign body is unattached at the point of entrance, the missile is transported through the vena cavae into the right auricle and through the tricuspid valve into the right ventricle. It is because of this rare condition that this case is presented. Harkins demonstrated in the last war the feasibility of the removal of these foreign bodies and the dangers encountered in leaving them *in situ*. Since that time, there have been many intracardiac procedures evolved for various acquired and congenital anomalies to make an exploration of the ventricles a reasonable procedure. With due application of the known surgical principles relative to cardiac surgery, such an operation can be performed with relative safety. An accurate preoperative diagnosis is essential in planning an appropriate incision to afford unhindered exposure. A small, but well-placed, incision in the myocardium, with considerable thought given to adequacy of incision and preservation of blood and, at the same time, preservation of the normal coronary vessels, is necessary. It is extremely important not to displace the heart from its normal position *in situ*. It is adherence to the latter principle which influences greatly the cardiac embarrassment which can easily follow cardiac manipulation. Procaine hydrochloride, 1%, sprayed directly on the myocardium reduces its sensitivity also. The incision into the pericardium must be adequate and may be made with relative impunity, extending from one phrenic nerve to the other. This exposes the entire anterior surface of the heart. Since it is not possible for the pericardial sac to tolerate collections of fluid which accumulate rapidly, causing cardiac tamponade, the pericardium must be drained. Since it is best not to drain it externally because of the danger of infection, the problem is best solved by draining into the pleural space, which in turn is easily drained through an underwater seal type of drainage. It must also be remembered that the pericardium must be partially closed because it is possible after operation for the heart to herniate through a pericardial incision and to produce distortion of the heart, with resulting extreme cardiac embarrassment. A drainage site of about 2 in. (5 cm.) would fulfill the requirements of safety and insure adequacy of drainage.

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REPORT OF A CASE

Private A. O. B., a 22-year-old white soldier, was admitted to the 8063d MASH Unit on Sept. 7, 1951, a few hours after sustaining injury when the truck on which he was riding hit an enemy land mine near Chorwon, North Korea. The patient sustained soft tissue wounds of both buttocks and the left thigh, right leg, and left arm. He had a débridement of his wounds and removal of several metallic foreign bodies from the lower extremities. On Sept. 14, his wounds were closed secondarily, and a routine chest x-ray revealed a foreign body lying within the chest. There were no external wounds of the chest or of the abdomen. Further examination by x-ray and fluoroscopy showed the foreign body, which measured about 1 cm. in diameter, to be lying either in the wall of the right ventricle or in the pericardium.

The patient was transferred to the Osaka Army Hospital on Oct. 25, 1951. When admitted there he had several well-healed wounds of both buttocks and of the left leg and forearm. There were no wounds of the chest or of the abdomen. Examination of the chest revealed no abnormalities. There was no evidence of any cardiac abnormality, and the patient apparently

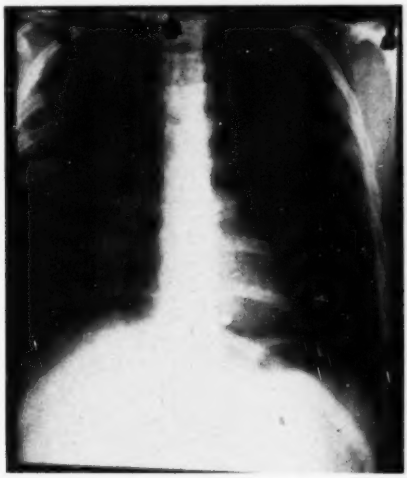

Fig. 1.—Posteroanterior view showing foreign body in right ventricle.

was in excellent physical condition. X-ray examination of the heart showed a normal cardiac silhouette and a foreign body. Posteroanterior, lateral, and oblique views showed the foreign body to lie either in the wall of the right ventricle, or in the pericardial cavity adjacent to the right ventricle, or possibly in the right ventricle itself (Figs. 1 and 2). The electrocardiogram revealed normal function. There was normal sinus rhythm, pulse rate of 72, no cardiac enlargement, and no thrills or murmurs. He had no complaints of cough, hemoptysis, chest pain, dyspnea, or cyanosis. The reaction to cardiolipin was negative. A complete blood count was normal, with a hemoglobin concentration of 15.8 gm. per 100 cc. and a hematocrit level of 46. Urinalysis revealed no abnormalities. Fluoroscopy confirmed the presence of a foreign body in the region of the right ventricle, and this foreign body moved with cardiac action. There was no evidence that the foreign body moved with changes in position.

On Nov. 5, 1951, an exploration of the pericardium and heart was performed, with the patient lying supine and with use of intratracheal ether anesthesia after induction with thiopental (pentothal®) sodium and curare. The incision was made extending from the midportion of the sternum laterally along the course of the sixth rib anteriorly to the anterior axillary line just below the nipple. The presenting portion of the sixth rib and its cartilage were resected subperiosteally and subchonderally. Portions of the cartilage of the lower ribs were resected

at the junction with the sternum, and a portion of the sternum itself was rongeured out for exposure. The internal mammary artery was isolated, doubly ligated, and cut. This incision exposed the pericardium up to the right auricle and as far laterally as 2 in. over the left pleural space. The left pleural cavity was deliberately opened and found free. There was no evidence of any external wound to either the pleura or the pericardium, and the heart was not enlarged. The pericardium was then opened transversely from just to the left of the right auricle over to the left phrenic nerve. There was no abnormal amount of fluid within this pericardial space, and there was no evidence of any trauma to the pericardial sac or to the myocardium itself. The surface of the heart was then bathed in about 5 cc. of 1% procaine hydrochloride. The incision gave adequate exposure to the entire anterior surface of the heart. In the sulcus between the right auricle and the right ventricle, just to the left of the right coronary artery, the foreign body could be palpated through the wall of the right ventricle. This was confirmed by means of the Berman locator. Two stay sutures were placed on the anterior surfaces of the right ventricle, 2.5 cm. apart, through the wall of the ventricle directly over the foreign body, with due care to preserve the right coronary artery and vein. An incision 1 cm. long was made in the longitudinal axis of the heart, care being taken to preserve the right coronary vessels. This

Fig. 2.—Lateral view showing foreign body in right ventricle.

incision was made through the entire thickness of the wall of the right ventricle. With slight tension of the stay sutures and with a finger placed over the aperture, only a small spurt of blood escaped on two occasions from the heart itself. A grasping forceps was inserted through the hole; the foreign body was located and removed without difficulty. It was only loosely attached to the endocardium. This foreign body was rather sharp on its edges and had adherent cloth attached and measured about 1 cm. in diameter (Figs. 3 and 4). The blood loss during this manipulation did not amount to more than 100 cc. The myocardium was sutured with interrupted 00 nonabsorbable surgical (black silk) sutures which did not enter the endocardium. The myocardium was again bathed with 1% procaine hydrochloride. There was no evidence of any cardiac irregularity throughout the procedure. The pericardium was sutured with interrupted black silk, leaving a 2-in. gap on the left side which drained into the previously made incision through the left pleural space. The left pleural space was then drained with a Foley catheter connected to an underwater seal drain. Procaine hydrochloride was again inserted into the pericardial sac. Penicillin, 200,000 units, and streptomycin, 1 gm., were injected into the pleural space and about the operative wound. The intercostal muscles were sutured with interrupted black silk and the wound closed in layers.

It was estimated that the loss of blood volume during the operation amounted to about 1,500 cc., which was replaced by whole blood. No other fluid was given. The patient was returned to his bed and placed in an oxygen tent. His blood pressure was maintained at a level, with a range of 100/70 to 130/80 throughout the immediate postoperative period. The pulse was rapid, 120 beats per minute, but was regular, and the heart sounds were clearly audible. There were no thrills or murmurs. An electrocardiogram taken one hour after surgery showed a

Fig. 3.—Metallic foreign body.

Fig. 4.—Photomicrograph of metallic foreign body removed, showing adherent cloth.

normal tracing. On the second postoperative day, the general condition remained excellent; there was no evidence of any cyanosis or dyspnea. X-ray study showed the lung to be well expanded, and drainage from the intercostal catheter amounted to about 100 cc. of serosanguineous material. The blood pressure at this time was 120/80. On the third postoperative day the intercostal tube was removed. The x-rays showed complete expansion of the lung, and the drainage had ceased. The patient's pulse rate was about 100 and was always regular; the heart sounds were clear, and no thrills or murmurs were heard. An electrocardiogram taken

on this day was interpreted as showing evidence of a generalized pericarditis but no evidence of localized myocardial necrosis. On the fourth postoperative day, the general condition was excellent; the heart rate began to return to about 90 and was regular, and the patient was allowed to dangle his legs. The patient was afebrile; the wound was clean, and the patient felt well. The electrocardiogram was about the same as the previous day. On the fifth postoperative day, the electrocardiogram was interpreted as still showing some pericarditis, but the pulse rate was returning to normal. By the sixth postoperative day, the electrocardiogram was returning to normal; the patient was ambulatory and had no complaints of dyspnea or of pain. The pulse was regular, ranging from 80 to 90 beats per minute. A repeat blood count showed 4,920,000 red cells, a hematocrit of 50%, and 16 gm. of hemoglobin per 100 cc., indicating that the patient probably did not lose as much blood as had been estimated. By two weeks after operation, the patient's general condition was excellent—he was walking around tne ward in no distress whatever. Physical examination of the heart suggested some right ventricle enlargement, but this was not confirmed by x-ray or fluoroscopy. The pulse rate remained regular, and the electrocardiogram continued to be normal, although there were some inverted T waves which were interpreted by the cardiologist as indicating a postoperative pericarditis. Three weeks postoperatively, the patient was walking around the ward; the electrocardiogram was normal, and he had no complaints.

COMMENTS

This case is thought to represent an embolic foreign body, proof of which, of course, is essentially indirect. There was no external wound of the chest wall, of the pericardium or of the myocardium, or of the diaphragm. The posterior surface of the heart, however, could not be examined completely. It was thought that a single foreign body, one out of many with which the patient was sprayed in the lower extremity, entered the iliac or femoral veins and thus gained access to the inferior vena cava. There was no evidence of any vascular damage in the lower extremity, either at the former military installations where the wounds were initially débrided or on physical examination at the time of admission here. It is entirely possible that such a phenomenon could occur. The exact location of this foreign body could not be ascertained preoperatively. It did not move with changes in position, and roentgenographically it could have been in the pericardium or in the myocardium.

Whether this foreign body would cause difficulty in the future, in view of its asymptomatic nature, was the question. Such a foreign body would necessarily constitute a distinct hazard as a potential pulmonary embolus, or, because it was contaminated (cloth was still adherent at the time of removal), it could be a source of multiple septic pulmonary emboli, with resulting lung abscess or septicemia. There was also distinct danger of a cardiac aneurysm forming because of the constant motion to which this sharp foreign body was subjected every minute.

To prevent cardiac irregularity, 1% procaine hydrochloride was used to bathe the heart at frequent intervals. Quinidine sulfate was not used in this case, although it was kept on hand for the postoperative course in case of arrhythmias. The heart was not disturbed from its bed in any way, and no stay sutures were placed through the apex. Special care was taken in the anesthesia to prevent anoxia which might have lowered the threshold to produce arrhythmia. Oxygen was given constantly postoperatively for two days in a tent and with nasal administration of oxygen to prevent this complication. With these measures in force, no irregularity developed.

The problem of treating postoperative cardiac tamponade was dealt with prophylactically by leaving a 2 in. vent in the pericardium to drain into the open pleural

space which could easily handle large amounts of fluid and which was much more easily and safely drained externally. The major portion of the pericardial incision was resutured rather firmly because of the postoperative danger of herniation.

The question of incision is rather an important one, in that exposure must fulfill the criteria of adequacy and postoperative physiological function (Fig. 5). Generally speaking the apex of the heart formed by the left ventricle lies just below the nipple in the left fifth intercostal space. The right ventricle projects on the anterior wall from the left half of the sternum to the left parasternal line about 2 cm. from the left border of the sternum between the third to the sixth intercostal cartilage level, forming most of the anterior surface of the heart. The left border of the cardiac silhouette is, of course, formed by the left ventricle. With this thought in mind, an incision which involves resection of a small portion of the sternum and the sixth costal cartilage and portion of the sixth rib should and did give complete and adequate exposure to the entire right ventricle (Fig. 5). As far as incision in the pericardium

Fig. 5.—Photograph showing incision for exposure of right ventricle.

is concerned, this is not too important in that there are no large vessels if one makes a transverse incision from one phrenic nerve to the other, and this produces adequate exposure of the entire anterior surface of the heart. The myocardial incision itself is considered in the light of the surgical procedure to be performed. Although in this case the approach might have been through the right auricle, it did not seem quite feasible because the foreign body was attached to the endocardium and apparently entwined in the chordae tendineae of the tricuspid valve. The ventricular approach was simple and direct and produced no physiological disturbance that was apparent. Due care must, of course, be exercised in the preservation of the coronary vessels and in the direction of the incision so as to prevent distal ischemia.

In regard to the blood-volume replacement in such a case, it was thought best to follow the general surgical principle of replacing the blood loss as it occurs. This seemed especially applicable to this type of case, in that it is reasonable that overloading of the vascular tree would be more conducive to cardiac failure. This patient had 1,500 cc. of blood replaced during the operation and during the immediate

postoperative period, which was probably more than adequate. In view of the radical nature of the operation, blood was started on two extremities, and although the loss was not great, the patient unavoidably received blood prophylactically. Saline solution was not administered because of the adequacy of replacement. The patient's postoperative course was not complicated by any particular pulmonary or vascular disturbance, except for his rapid, although regular, pulse.

SUMMARY

This is a case of a young soldier who was involved in the explosion of a land mine and sustained soft tissue wounds of the extremities and an embolic metallic foreign body which migrated to the right ventricle of the heart. This patient was treated surgically, with successful removal of the foreign body, and has no postoperative residua. The surgical physiology of the principles are discussed with reference to their particular application to this case.

PANCREATIC CYST: AN UNUSUAL CAUSE OF HYPERSPLENISM

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HYPERSPLENISM, first described by Wiseman and Doan,[1] includes a variety of hematologic syndromes which can be attributed to overactivity of the spleen. The spleen has three major functions: the destruction of red blood cells, the storage of blood, and the production of lymphocytes from the lymphoid elements in the organ. In hypersplenism, there may exist a diminution in any of the cellular elements of the blood. The patient may exhibit findings consistent with neutropenia, thrombocytopenia, or anemia, either singly, or in combination, or all three together.[2] The etiologic factors enumerated by Dameshek[3] in a study of 28 cases included rheumatoid arthritis, syphilis, cirrhosis of the liver, Gaucher's disease, Boeck's sarcoid, malaria, and Hodgkin's disease, and 16 were classified as idiopathic. The diagnosis is based on the cytopenia associated with a normal or hyperfunctioning bone marrow, and in the case of anemia, with no obvious cause of blood loss. The cardinal features of the disease include: (1) an enlarged spleen (except in thrombocytopenic purpura); (2) cytopenia; (3) normal or hyperfunctioning bone marrow, and (4) demonstration of splenic overactivity by the epinephrine test. There are two theories of the pathogenesis of the hypersplenic state: (1) phagocytosis of the cellular elements in the spleen,[1] and (2) inhibition of maturation of the cellular elements of the marrow by an agent liberated by the spleen.[3] Prognosis without splenectomy is a slow downhill course. The cure rate after splenectomy is 80%.

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The following patient, who exhibited splenomegaly, diminution in red cells, and a hyperplastic bone marrow, had a cyst in the tail of the pancreas eroding the hilus of the spleen as a cause of hypersplenism.

REPORT OF A CASE

A 40-year-old, widowed, white man was admitted to the hospital on April 18, 1951, with the chief complaint of severe epigastric and left upper quadrant pain of three months' duration. After his wife's untimely death in December, 1948, the patient drank large amounts of whiskey and wine and was in an almost constant state of inebriation. In January, 1951, he vomited an estimated quart of blood, which was followed by melena of two days' duration. After the hematemesis, the patient had attacks of severe epigastric and left upper quadrant pain which radiated to the back and caused him to double up. The severe pain lasted two days and was followed by residual soreness in the left upper quadrant for several more days. These attacks occurred every 7 to 10 days and occasionally were accompanied by nausea and vomiting. During the four months before admission, he noted increasing shortness of breath, weakness, and intermittent swelling of the right ankle. He had lost 27 lb. (12.2 kg.) during the three years prior to admission.

Physical examination revealed a pale, ill-appearing patient who tended to be irritable. The liver was palpated two fingerbreadths below the right costal margin. In the left upper quadrant a tender mass was palpated which extended two fingerbreadths below the left costal margin. Slight muscle spasm was observed in this area. The mass did not descend on respiration, nor could its upper border be delineated.

Examination of the blood revealed a hemoglobin concentration of 4.6 gm. per 100 cc., a red cell count of 2,500,000, and a white cell count of 9,900 on admission. The sulfobromophthalein test showed a retention of 2.5%; the icteric index was 5 units, and the prothrombin time was 100%. Thymol turbidity was 0.9 units and alkaline phosphatase 3.8 units. Of five stool specimens examined, only one gave a + + reaction for occult blood in the guaiac test. The blood sugars, chlorides, carbon-dioxide-combining power, and calcium and phosphorus determinations were within normal limits. Clot retraction and red cell fragility, as well as bleeding and clotting times, were within normal limits. The oral glucose tolerance test revealed a fasting level of 100 mg. per 100 cc.; 190 mg. per 100 cc. one-half hour later; 248 mg. per 100 cc. one hour later, and 233 mg. per 100 cc. two hours later, at which time there was a trace of sugar in the urine. Successive amylase tests were read as 90, 175, 161, 132, 80, 85, and 75 units (normal upper limit, 250 units). Simultaneous lipase determinations were 0.8, 2.9, 1.6, 1.3, 0.2, 0.3, and 0.2 units (normal upper limit, 1.0 unit). Eight units of free hydrochloric acid were found in the gastric fluid specimen. The urine was alkaline, with a specific gravity of 1.020, and contained a trace of albumin and 0 to 1 white blood cells per high power field. One of four urine specimens contained 2 to 3 red blood cells per high power field. A phenolsulfonphthalein test showed 30% excretion in 15 minutes. Reactions to the Mazzini and Coombs tests were negative. Bone marrow smears on April 6, 1951, gave the following differential count: myeloblasts 1, myelocytes 18, metamyelocytes 31, neutrophilic granulocytes 18, eosinophilic granulocytes 1, basophilic granulocytes 1, lymphocytes 29, reticulum cells 1, and normoblasts 27/100 white cells. Megakaryocytes and platelets were present in adequate numbers.

X-Ray Studies.—An intravenous pyelogram revealed downward displacement of the left kidney. Perineal air insufflation showed the left kidney lying lower than the right. About the left kidney there was seen a relatively homogeneous density which seemed intimately connected with the spleen. The diagnosis was a splenic or pancreatic cyst. The gastrointestinal series revealed slight widening of the loop of the second portion of the duodenum. At a later examination, after the stomach was distended with barium and air, a filling defect of the posterior wall measuring 8 cm. in length was demonstrated. This finding was consistent with a large retrogastric mass.

Clinical Course.—On the second hospital day the patient complained of severe left upper quadrant pain, radiating to the back. The pain lasted five hours and was followed by residual soreness. On the following day shaking chills and fever to 102 F. developed, which lasted 24 hours. Attacks of left upper quadrant pain recurred daily and were especially pronounced after eating. He required opiates with increasing frequency.

Although he was given 3,000 cc. of blood within the first three days of hospitalization, the hemoglobin level could not be maintained at normal values. He required an additional 1,500 cc. of blood on May 22, 1951, after which the hemoglobin rose from 9.5 to 12.5 gm. but dropped again to 10.5 gm. prior to operation.

At operation on June 11, 1951, a long, oblique, upper abdominal incision traversing both rectus muscles was made. In the tail of the pancreas there was a moderately firm mass about 3 in. (7.5 cm.) in diameter, which was intimately adherent to the spleen and to the posterior wall of the stomach. The head of the pancreas was moderately thickened. The spleen was almost three times normal size and somewhat inaccessible. Accordingly, the abdominal incision was extended into the left eighth interspace, and the diaphragm was opened. The spleen was freed

Cut surface of enlarged spleen can be seen on the left. In upper right-hand corner, ragged wall of pancreatic cyst can be observed. Below this structure lies thickened indurated pancreas containing markedly sclerosed vessels.

from all its attachments. The cyst was opened to facilitate its dissection from the muscular layer of the stomach. It contained about 100 cc. of soft, dark blood clots and material that resembled splenic pulp. The tail of the pancreas was freed and divided. The spleen and tail of the pancreas, which included the cyst, were removed. The cut end of the pancreas was reapproximated with nonabsorbable surgical (silk) mattress sutures and covered by a portion of omentum, which was sutured in place. A small incision was made in the left upper quadrant through which a sump drain and a penrose drain, placed near the cut end of the pancreas, were withdrawn. A catheter was placed in the pleural space. He made an uneventful recovery and was discharged on the 15th postoperative day. At the time of discharge, the hemoglobin was 13.5 gm. per 100 cc., the platelets 68,000, and the white cell count 8,050. He was asymptomatic when examined eight months later.

Pathologic Examination.—The specimen was a 260 gm. spleen measuring 11 by 7 by 5.5 cm. (Figure). The capsule was diffusely and irregularly thickened and covered with numerous vascular adipose adhesions. The hilum was replaced by a cavity 2 cm. deep and 7 by 3.5 cm. in diameter. The cavity was lined by a shaggy, yellow-brown membrane 1 mm. thick, and it contained 5 cc. of dark red blood clot. Its wall measured from 1 mm. to 2 cm. in thickness and appeared to consist of fibrofatty tissue, traversed by the stenotic hilar vessels of the spleen. At the inferior posterior pole of the spleen, just beneath the capsule, there were two firm, sharply demarcated, mottled pink and yellow areas of roughly triangular shape, each measuring 1 cm. in diameter. The rest of the pulp was firm and pale red, with scanty scrapings and inconspicuous follicles. Microscopic section revealed two relatively fresh infarcts with peripheral hemorrhage and marked thickening of sinusoidal walls in their vicinity. A small vessel adjacent to an infarct contained a fresh thrombus. An occasional megakaryocyte was found within the sinusoids. The cyst wall was lined by hemorrhagic and fibrinoid material, the outer portion of which was organizing and intimately associated with the hilum of the spleen. A great deal of blood pigment was deposited in these areas. Sections through the thickest portion of the cyst wall showed fibrotic pancreatic tissue in which all parenchyma was eradicated, except for a few islets. The arteries showed marked intimal proliferation with narrowing of lumina. Chronic inflammatory cells were scattered through the fibrous tissue and about the blood vessels.

Diagnosis.—The diagnosis was chronic pancreatitis with marked pancreatic fibrosis, pancreatic pseudocyst, mild splenomegaly, and infarcts of the spleen.

COMMENT

Although this patient showed no evidence of bleeding after hospital admission, it was impossible to maintain his hemoglobin at normal levels despite the administration of multiple transfusions before operation. The diagnosis of hypersplenism was made on the basis of refractory anemia, splenomegaly, normoblastic hyperplasia of marrow, and maintenance of normal hemoglobin levels following splenectomy without necessity for further transfusions. A pancreatic cyst involving the hilum of the spleen and splenic vessels is postulated as the etiologic factor in the production of the hypersplenic state in this case. The excessive alcohol intake may have been responsible for the development of pancreatitis and secondary pseudocyst formation.

REGIONAL COLITIS CONFINED TO SIGMOID COLON

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THE CLASSIC appearance of ulcerative colitis in which the involvement of the rectum or rectosigmoid is succeeded by a more or less diffuse involvement of the colon is a familiar clinical problem. However, the occurrence of regional or segmental types is much less common and may present diagnostic difficulties. The least common of the segmental distributions is that in which the sigmoid alone is involved. Since the pathogenesis of this lesion is obscure and the diagnosis often difficult, it has seemed worth while to report a single case.

REPORT OF A CASE

J. K., a 34-year-old white man, was admitted to the Veterans Administration Hospital, Aug. 29, 1950, complaining of mild lower abdominal pain, weakness, malaise, and weight loss of six weeks' duration. There was no disturbance in bowel function and no change in the color or consistency of the stools. The patient appeared chronically ill and pale and thin, and he had a mild fever. The abdomen was flat and soft. There was a slightly tender, firm mass about 3 in. (7.6 cm.) in diameter palpable in the left lower quadrant of the abdomen, and this was thought to be the sigmoid colon. There was no tenderness on rectal examination. The diagnosis on admission was diverticulitis of the sigmoid colon with abscess formation. It was realized that carcinoma of the colon was also a possibility.

Course in the Hospital.—Initially the patient was very ill, with an irregular fever and moderate anemia and leucocytosis. Treatment included a nonresidue diet, liquid petrolatum given orally, penicillin injected intramuscularly, and blood transfusions. He slowly improved markedly with this treatment. The abdominal mass slowly decreased in size and eventually became a firm, fixed, elongated mass. Sigmoidoscopic examination gave normal results. Repeated examinations of feces showed no blood, ova, or parasites. Roentgenologic study of the colon by barium enema revealed a narrowing of the lumen of the sigmoid colon. No diverticula were seen. Because carcinoma had not been eliminated as a diagnostic possibility, a celiotomy was done on Sept. 26, 1950. A large inflammatory mass 12 cm. long was found involving the sigmoid colon and its mesentery, with dense inflammatory adhesions to the bladder. No diverticula were seen. A loop transverse colostomy was made, deferring resection of the diseased colon to a later date. The patient improved after the operation, although the roentgenologic appearance of the sigmoid colon did not change (Fig. 1). The diseased sigmoid colon was resected on Jan. 9, 1951, and an

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end-to-end anastomosis was done. At a later date, the transverse colostomy was closed. The patient remained asymptomatic for one year, and a barium enema at that time revealed a normal colon.

Report on Surgical Specimen.—The resected portion of the sigmoid colon was about 12 cm. long and 3 cm. in thickness. The opened specimen presented a marked narrowing except at the

Fig. 1.—Photograph showing resected sigmoid colon.

ends (Fig. 2). The central portion was markedly narrowed, measuring 1.5 cm. in circumference. At the ends the lumen was normal. The mucosa was edematous and hyperemic, and slightly polypoid in areas, but no ulcerations were present. The wall of the colon was thickened, measuring 8 cm. in the narrowed portion. Microscopic examination revealed chronic nonspecific inflammatory changes. The pathologic diagnosis was healed chronic ulcerative colitis.

COMMENT

Even after the diagnosis of localized ulcerative colitis is established, two problems remain: 1. What relation does this have to the diffuse form of ulcerative colitis? 2. What is the best form of treatment? Sloan, Bargen, and Gage [1] reported a study of 2,000 cases of ulcerative colitis. The involvement was segmental in 128 cases, the sigmoid colon alone being involved in 9 instances. Crohn, Garlock, and Yarnis [2] have recognized a form of ulcerative colitis involving the right colon which has progressed to the classic form of the disease. This right-sided colitis clinically

Fig. 2.—Roentgenogram showing diseased sigmoid colon.

and pathologically resembles diffuse ulcerative colitis more closely than does localized disease in the left colon. Other authors [3] have reported instances of ulcerative colitis involving the sigmoid alone, and these patients with chronic localized colitis

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do not have the classic picture of ulcerative colitis. Fever, loss of weight, and vague abdominal pain which is sometimes crampy have been outstanding symptoms. Tenesmus, diarrhea, and bleeding have often been absent. Usually there is considerable narrowing of the colon in the area involved. Obstruction [4] and perforation with abscess formation may occur.

Recently, Lumb [3b] has suggested that these cases are really more closely related to regional enteritis than to ulcerative colitis and has pointed to the many similarities in gross and microscopic pathology. Warren and Sommers,[5] however, reported that a differential diagnosis is possible, as did Rappaport and co-workers.[6] The absence of granulomatous lesions, the less marked submucosal edema, and the less marked fibrotic process are features which influence the latter authors to differentiate this lesion from regional enteritis.

The treatment of sigmoidal colitis appears to be most satisfactory. After initial supportive treatment, resection is indicated; occasionally, a preliminary transverse colostomy may be necessary. The results of local resection in reported cases indicate that there is little chance of recurrence or spread.[7]

SUMMARY

A case of regional colitis is presented in which a good result was secured by resection.

The differences in clinical and pathologic behavior of the regional disease from the more diffuse type of chronic ulcerative colitis have been stressed.

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COEXISTING PEPTIC ULCER AND RETICULUM CELL SARCOMA OF THE STOMACH

C. ANTONIO DOHSE, M.D.
AND
RODERICK C. ROSS, M.D., M.Sc.
MONTREAL, CANADA

A CASE is presented in which gastric resection revealed the coexistence of chronic peptic ulcer and reticulum cell sarcoma of the stomach.

Reticulum cell sarcoma of the stomach is a relatively rare lesion. In 1,220 cases in which malignant gastric neoplasm was seen at the Cleveland Clinic, Spencer, Collins, and Renshaw [1] found only 4 cases of reticulum cell sarcoma, an incidence of 0.3%. In 18,668 autopsies made at the Boston City Hospital, 28 cases of reticulum cell sarcoma were found, of which 4 were primary in the stomach.[2]

The occurrence of sarcoma in association with chronic peptic ulcer is of interest in view of the fact that some authors have suggested a relationship between the two lesions.

REPORT OF A CASE

A 73-year-old white woman was admitted to St. Mary's Hospital, Oct. 9, 1951, with a three-month history of continuous epigastric pain associated with vomiting. Her past history disclosed that she had suffered from occasional attacks of "indigestion" during the past seven or eight years, at intervals of two to six months, usually following dietary indiscretions. In July, 1951, after a holiday away from home, the epigastric pain became continuous and was unrelieved by the usual home remedies. She sought medical advice, and under an ulcer regimen she improved for seven or eight weeks. Early in September, 1951, the pain recurred with nausea and vomiting, and because of its persistence and severity she was admitted to the hospital. At this time the pain was continuous and radiated to the hypochondrium, chiefly on the left side and also to the back between the shoulders. She complained of constipation requiring the use of laxatives. There was no history of hematemesis or melena.

The family history revealed that her mother had suffered from gastric ulcers, but there was no history of cancer. Her past history revealed that she had had acute appendicitis and peritonitis in 1910.

Physical examination revealed a well-nourished white woman whose appearance corresponded with her age. There was no lymphadenopathy. The abdomen was obese, and there was rigidity and tenderness to deep palpation in the epigastrium and left upper abdomen. The only palpable structure was the ascending colon. There was no evidence of free fluid in the peritoneal cavity.

Examination of the blood revealed 4,110,000 red cells and 6,900 white cells per cubic millimeter. The concentration of hemoglobin was 12.1 gm. per 100 cc. There were 6.4 gm. of protein per 100 cc., of which 4.0 gm. were albumin and 2.4 gm. globulin.

From the Department of Pathology, St. Mary's Hospital.

1. Spencer, F. M.; Collins, E. N., and Renshaw, R. J. F.: Sarcoma of Stomach, Cleveland Clin. Quart. **14**:282-295, 1947.

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Fig. 1.—X-rays of the stomach showing two well-defined ulcer craters on the lesser curvature.

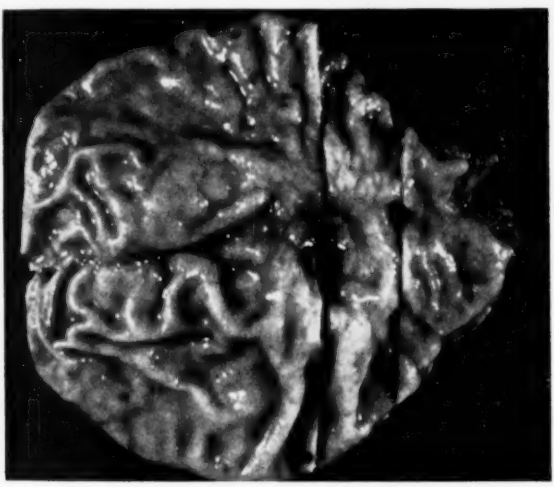

Fig. 2.—Gross appearance of the stomach after fixation with formaldehyde. A 4 mm. section through each lesion has been removed. The section on the left passes through the peptic ulcer, the one on the right through the reticulum cell sarcoma.

X-ray examination showed a large deformity midway along the lesser curvature, with a smaller one approximately 1 in. (2.5 cm.) distal to it. These were interpreted as multiple gastric ulcers (Fig. 1).

With the patient under spinal anesthesia a subtotal gastrectomy was performed. The postoperative course was uneventful, and she was discharged on the 12th postoperative day. The patient has remained asymptomatic.

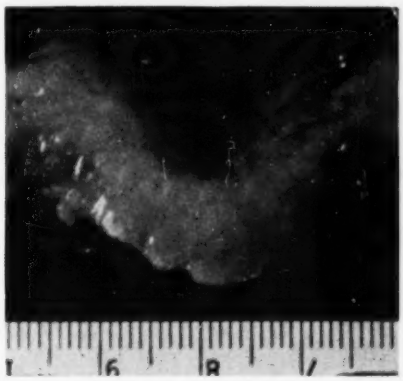

Fig. 3.—Cross section through the peptic ulcer.

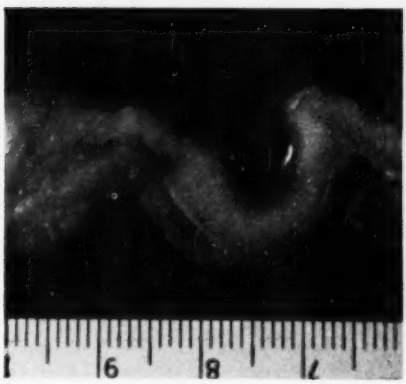

Fig. 4.—Cross section through the reticulum cell sarcoma.

PATHOLOGICAL FINDINGS

Gross Description.—The operative specimen consisted of the distal portion of a stomach measuring 15 cm. along the greater curvature and 10 cm. in the maximum transverse dimension. There was a 20 by 18 cm. segment of omentum attached to the greater curvature. The fatty tissue on the lesser curvature was indurated over an area 5 by 3 cm.

Opening the stomach revealed on the lesser curvature an ulcer 3.5 by 3 cm. located 3.5 cm. from the distal margin of resection. This ulcer was approximately 1 cm. in depth, had a shelving margin and light grey base. Midway between it and the pyloric margin there was a smaller, shallow ulcer 1 cm. in diameter (Fig. 2).

Fig. 5.—The main reticulum cell sarcoma is on the left, and the floor of the peptic ulcer is parallel to the right margin of the photograph. An additional focus of reticulum cell sarcoma is evident at the margin of the peptic ulcer. Penetration of the muscularis by the peptic ulcer is evident; × 10.

Section through the larger ulcer revealed it to be a typical chronic peptic ulcer which had destroyed the muscle and penetrated the adjoining fatty tissue. Its base was composed of firm, fibrous tissue 1 cm. in thickness (Fig. 3). Section through the smaller ulcer revealed its base to be a cup-shaped mass of glistening white tissue 2 cm. in diameter and superficial to the muscular coat (Fig. 4). Sections of the

stomach in the long axis through the center of both ulcers showed that the two lesions were separate. However, in the adjacent margin of the peptic ulcer there was a smaller focus of the same glistening white tissue which composed the distal lesion (Fig. 5).

One lymph node 1 by 0.8 by 0.5 cm. was found on the lesser curvature. Several small lymph nodes were present in the greater omentum.

Microscopic Findings.—Histological sections of the upper lesion in the transverse plane revealed a typical chronic peptic ulcer. The mucosa was very slightly overhanging, and there was some marginal reepithelization but no evidence of epithelial atypism. The ulcer had penetrated the entire muscularis, and its base was composed of granulation and collagenous tissue. One artery of moderate size in the floor of the ulcer showed erosion of the wall and thrombosis. One section in the same plane showed at the margin of the ulcer a small focus of reticulum cell sarcoma.

Microscopic sections of the ulcerated lesion in the distal portion of the stomach revealed reticulum cell sarcoma confined to the mucosa and submucosa. There was one main lesion and smaller foci immediately adjoining the parent lesion. All the lesions lacked peripheral demarcation. The gastric glands were preserved to a variable degree, and in the central portion of the lesion the mucosa was ulcerated.

Histological sections in the longitudinal axis through the centers of the two lesions revealed ulcerating reticulum cell sarcoma on one side, a short interval of normal tissue, then an additional focus of reticulum cell sarcoma constituting one margin of the peptic ulcer (Fig. 5).

Sections of the tumor stained for reticulum by Wilder's technique revealed a variable amount of reticulum which only occasionally encompassed individual cells. Mucicarmine and periodic acid-Schiff stains failed to reveal any evidence of mucin in the tumor.

Sections of the lymph nodes showed only slight hyperplasia of the reticulum cells in the germinal centers of the lymphoid follicles.

COMMENT

It would seem probable that the large, chronic peptic gastric ulcer was the initial lesion, and its structure correlates with symptoms of several years' duration. The reticulum cell sarcoma appears to be an independent lesion and the occurrence of foci of tumor at the margins of the peptic ulcer is probably merely a result of its multicentric origin.

Several authors [3] have suggested a possible relationship between gastric ulcer and sarcoma but have failed to produce convincing evidence. Marshall and Meissner [4] (1950) in a review of 41 cases of gastric sarcoma found nothing to suggest

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a relationship between peptic ulcer and sarcoma. Rabinovitch, Pines, and Grayzel [5] (1952) reported the coexistence of gastric lymphosarcoma and a peptic ulcer. Carcinoma had developed in the margins of the peptic ulcer, but the lymphosarcoma appeared to be an unrelated lesion. In our case the two lesions were in intimate relationship, but we believe that this was fortuitous.

SUMMARY

A case of coexistent chronic peptic ulcer and reticulum cell sarcoma of the stomach is presented.

The reticulum cell sarcoma was multicentric in origin, and although foci were found in the margin of the peptic ulcer, it is believed that this was fortuitous.

In a brief review of the literature no case was found with conclusive evidence of a relationship between peptic ulcer and gastric sarcoma.

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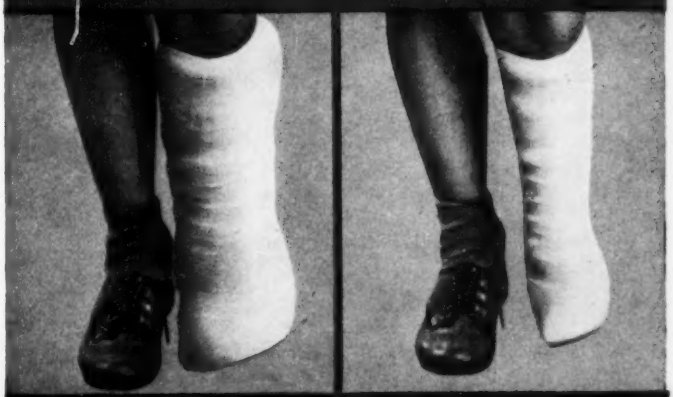

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1. A. W. Spittler, Col., (M.C.), U.S.A., J. J. Brennan, Lt. Col., (M.C.), U.S.A., J. W. Payne, Capt., U.S.A.F. (M.C.), American Academy of Orthopedic Surgeons, Jan. 26-31, 1952, Chicago, Illinois.
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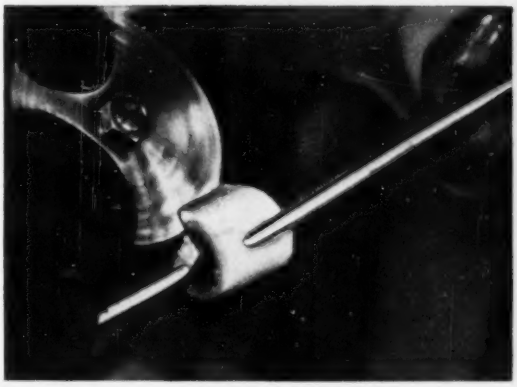

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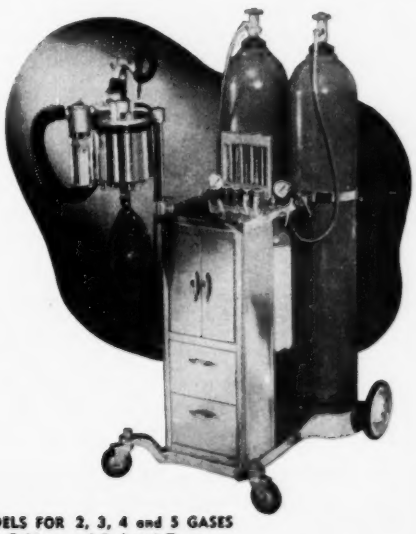

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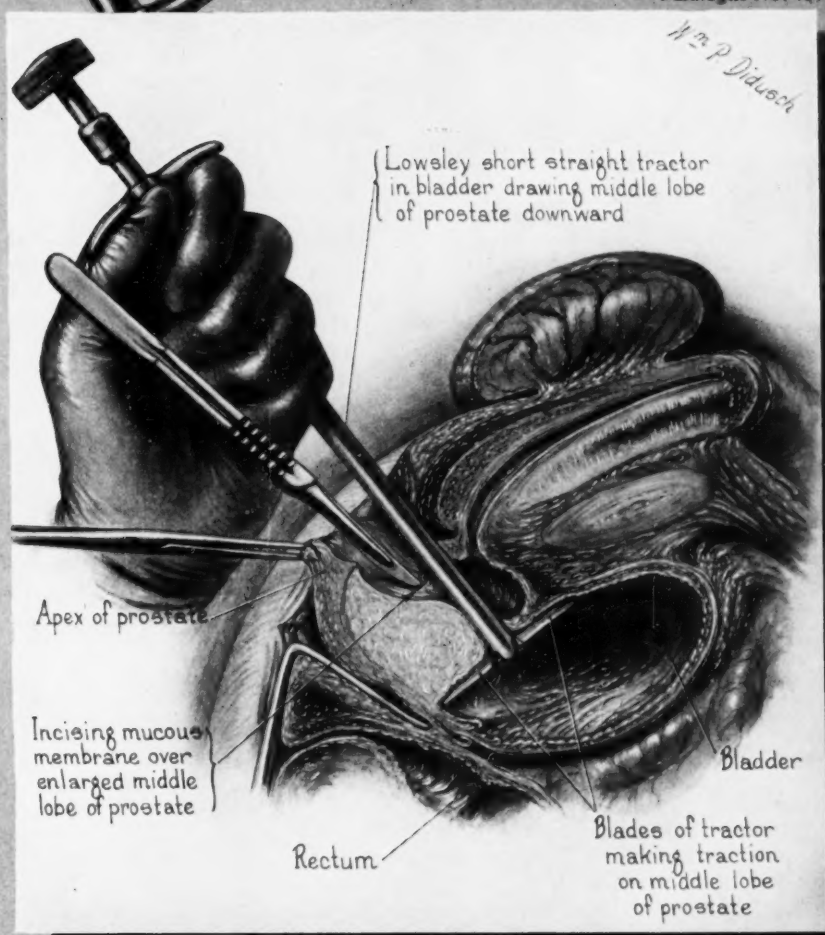
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